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Thermoregulatory Consequences of Long-Term Microwave Exposure at Controlled Ambient Temperatures

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by

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16. ABSTRACT

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ABSTRACT

This study was designed to identify and measure changes in thermoregulatory response systems, both behavioral and physiological, that may occur when squirrel monkeys are exposed to 2450-MHz CW microwaves 40 hours/week for 15 weeks. Microwave power densities explored were 1 and 5 mW/cm^2 (SAR=0.16 W/kg per mW/cm^2) and were presented at controlled environmental temperatures of 25, 30, and 35 °C. Standardized tests, conducted periodically, assessed changes in thermoregulatory responses. Dependent variables measured included body mass, certain blood properties, metabolic heat production, sweating, skin temperatures, deep body temperature, and behavioral responses by which the monkeys selected a preferred environmental temperature. Results showed no alteration of metabolic rate, internal body temperature, or thermoregulatory behavior by microwave exposure although the ambient temperature prevailing during chronic exposure could exert an effect. An increase in sweating rate occurred in the 35 °C environment, not enhanced significantly by microwave exposure. Skin temperature, reflecting vasomotor state, was reliably influenced by both ambient temperature and microwaves. The most robust consequence of microwave exposure was a reduction in body mass which appeared to be a function of microwave power density.

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INTRODUCTION

It is generally agreed that insufficient data exist to properly evaluate the biological consequences of long-term exposure to sources of nonionizing radiation. Committees of both houses of the Congress (U.S. Senate, 1978; U.S. House of Representatives, 1979) have urged the establishment of research programs which would investigate the biological effects of long-term exposure to low-level radiation. Held to be of particular importance is "...basic research into the physiological mechanisms by which biological and/or behavioral effects of exposure to nonionizing radiation may occur..." (U.S. Senate, op. cit., p 5). The present report describes some results of a four-year program of research for which the above description is highly appropriate. Specifically, we investigated the changes in thermoregulatory mechanisms that may occur when squirrel monkeys are chronically exposed to low-level microwave fields at controlled environmental temperatures.

Thermoregulation refers to the ability of an organism to achieve and maintain a characteristic internal body temperature. Two classes of responses can accomplish thermoregulation, behavioral and physiological (or autonomic). Behavioral responses, voluntary actions by the organism, control the thermal characteristics of the air-skin interface and can take two forms, either direct manipulation of the environment itself or bodily movement into a more favorable microclimate (Cabanac, 1983). The heat exchange between the organism and the environment is thereby closely regulated, which results in stability of the internal body temperature. Both types of behavior have been shown to change when an organism is exposed briefly to a microwave field (Adair and Adams, 1980b; Gordon, 1983; Stern, et al., 1979).

Physiological responses are involuntary in endothermic species and may occur automatically whenever behavioral responses are impossible or inadequate, or under special circumstances such as during exercise or febrile disease. Specific physiological responses include shivering and non-shivering thermogenesis, changes in peripheral vasomotor tone, sweating and/or panting. These responses also have been shown to change when endotherms are exposed briefly to a microwave field (Adair and Adams, 1980a, 1982; Ho and Edwards, 1977; Phillips, et al., 1975).

That long-term exposure to low-level microwave fields may alter thermoregulatory function is suggested by some of the Eastern European clinical literature. A neurocirculatory syndrome involving cardiac, neural, and metabolic function was often found in Soviet workers who had labored in close proximity to microwave sources for 10 years or more (Baranski and Czerski, 1976; Sadcikova, 1974). Marha (1971) described long-term alterations in body weight, blood pressure, body temperature, and certain conditioned reflexes. Baranski and Edelwejn (1975) reported that workers exposed to microwaves for more than 10 years are copious sweaters compared to short-term workers. However, Edelwejn and his colleagues (1974) have pointed out that in such clinical studies average microwave exposures are estimated, proper dosimetry is non-existent, and other relevant environmental factors, such as temperature and humidity, are usually neither assessed nor controlled.

Laboratory investigation of the effects of long-term microwave exposure in animals has been carried out almost exclusively on small mammals (i.e. mice, rats, guinea pigs, and rabbits). To date, no published study has stated as a specific purpose the assessment of the thermoregulatory consequences of such exposure, although a metabolic compensation in exposed animals has been inferred from measurements of body mass and food consumption. In general, rats and rabbits exposed for several weeks or months to low doses of microwaves eat less than controls but maintain body weight and increase fluid intake (Ferri and Hagan, 1975; Lovely, et al., 1977; Moe, et al., 1976). Djordjević, et al. (1977) reported a slight weight gain in both experimental and control rats exposed at 5 mW/cm^2, 1 h/day for 90 days. Lovely, et al., (1983) observed a reduced total food intake in rats exposed to 915- and 2450-MHz microwaves at 5 mW/cm^2 relative to sham-exposed controls, but no significant difference in body mass between the two groups across 134 days of exposure. They concluded that the exposed animals had profited from the microwave energy and the reduced energy demands of their bodies was reflected by a lowered food intake. Recently, Chou, et al. (1983) confirmed this finding in rabbits chronically-exposed for 90 days to 2450-MHz CW microwaves at 5 mW/cm^2; however, only food consumption during the 7-h daily exposure was measured, not 24-h intake. On the other hand, D'Andrea, et al. (1979) found no significant effects of microwave irradiation on either the food and water intake or the body mass of rats similarly exposed. During the 25-month chronic exposure study at the University of Washington (Guy, et al., 1980), periodic measurements of oxygen consumption and carbon dioxide production were made on selected members of the exposed and sham-exposed populations. No apparent metabolic changes occurred during the course of the chronic exposure (Guy, personal communication).

The effects of periodic and chronic microwave exposure at high power densities on the regulated body temperature of dogs was an early topic of investigation by Michaelson and his colleagues. Repeated exposure to

2800-MHz pulsed microwaves at the same intensity (165 MW/cm^2) produced smaller and smaller increases in rectal temperature (Michaelson, 1974). Also reported (Howland, et al., 1961; Michaelson, et al, 1963) was an adaptation to the procedure that took the form of a lower-than-normal body temperature, a result also found by Deichman, et al. (1963). Houk, et al. (1973) confirmed these results in rats chronically-exposed to 2450-MHz microwave fields at various power densities and suggested that the mechanism responsible might involve a resetting of the hypothalamic set point. Prausnitz and Süsskind (1962) suggested that the increased longevity of their microwave-exposed mice might reflect increased tolerance to febrile disease, conferred by daily "mini-fevers" which were artificially produced. A very important variable appears to be the ambient temperature at which such chronic exposures are performed, high ambients emphasizing thermoregulatory abnormalities and low ambients de-emphasizing them (Michaelson, et al., 1961; Mumford, 1969; Nikonova and Sokolova, 1974).

Few primates have been chronically exposed to radiofrequency fields and none for the express purpose of measuring changes in thermoregulatory function. McAfee, et al. (1979) have reported a continuing study at the Delta Regional Primate Center in which rhesus monkeys, living outdoors in large family groups, irradiate themselves with 9.3 GHz pulsed microwaves at a range of power densities as they work daily for apple juice rewards. No deleterious effects on the eyes have been observed after several years, and operant responding, social behavior, health, and fecundity are all normal. Grissett (1979) exposed rhesus monkeys for 22 months to extremely low-frequency electromagnetic fields but found no significant changes in blood chemistry, metabolic processes, or general health; the major difference between exposed and control animals was an increased growth rate in the former.

There is a clear need for basic information on the thermoregulatory consequences of long-term low-level exposure of primates to microwaves, preferably at different ambient temperatures. This paper describes a longitudinal study of highly trained animals (initially naive to microwaves) that combined both physiological and behavioral tests. Following the assessment of baseline levels of thermoregulatory function, squirrel monkeys were either sham-exposed or exposed to 2450-MHz continuous wave (CW) microwaves, 40 h/wk for 15 weeks. Power densities were 1 or 5 mW/cm^2 at controlled ambient temperatures of 25, 30, or 35 °C. Periodic physiological and behavioral tests were performed to assess thermoregulatory function during and after chronic exposure. The purpose of this study was to determine:

1) whether this exposure alters the normally-preferred ambient temperature and the ambient temperature selected during brief exposures to microwaves of higher field strength;

2) whether this exposure can produce "irritability" as measured by changes in the pattern of thermoregulatory responding;

3) whether significant changes occur in body mass and in standard hematological parameters;

4) whether this exposure changes the setpoint for body temperature, alters the normal physiological responses of heat production and heat loss in cold, neutral, and warm environments, or alters vasomotor activity; and

5) whether ambient temperature and microwave energy interact synergistically to produce changes in thermoregulatory function.

METHODS

1. Subjects:

Fourteen adult male squirrel monkeys (Saimiri sciureus) served as subjects. All but two (NIH-born) were feral-born in Peru or British Guiana. Their estimated ages ranged from 4 to 7 yr and their body masses ranged from 764 to 1038 g at the start of chronic exposure. Every monkey was experimentally naive to microwaves when first introduced to the project. During chronic microwave or sham exposure, animals were housed individually in cylindrical Plexiglas cages; during the physiological and behavioral tests, individual animals were restrained in a Plexiglas chair (Adair, 1969). Between exposures and experimental tests, the monkeys were caged individually in a vivarium held at 25 ± 2 °C with ad lib access to water. A daily ration of Purina monkey chow, fruit and vegetable supplements, and milk was fed in the home cage after completion of the day's experimental treatment. Animals were weighed every Wednesday at 0800 h.

Each animal selected for this study was highly trained to sit quietly in the restraining chair and to select a preferred environmental temperature behaviorally. The conditioning and training regime (Adair, et al., 1970) required a minimum of two years. In order to utilize a minimal number of monkeys while maintaining both näiveté to microwaves and the highest possible level of conditioning and training, each pair of sham-exposed animals for a given group became the microwave-exposed animals of the succeeding group. Thus, for example, the sham-exposed animals of Group III became the microwave-exposed animals of Group IV. Two to three months elapsed between groups to ensure the dissipation of any residual effects of the ambient temperature to which the animals had previously been exposed.

2. Chronic Exposure System:

a. Anechoic Chamber and Microwave Source

The system for chronic exposure included climate conditioning for the chambers housing both microwave-exposed and sham-exposed animals. Figure 1 shows a schematic diagram, as viewed from above, of the basic elements in this system. The anechoic chamber consisted of an electromagnetically-shielded room (Ray-Proof Corp., Norwalk, CT) of interior dimensions 2.45 x 2.45 x 3.66 m lined with 20-cm pyramidal microwave absorber (Advanced Absorber Products, AAP-8). A 1.83 x 2.45 m floor section at the rear of the chamber was covered with special flooring material (AAP-FL8); a climate-conditioned Styrofoam chamber which housed two animals was located in this area.

A 15-db standard gain horn antenna (Waveline Model 299) was mounted in the center of the transmitting wall of the chamber and connected to a 1 KW source of 2450-MHz CW microwaves (Cober Electronics, Model S1 3278) via standard waveguide. The electric field vector was aligned with the vertical dimension of the chamber. The power supply to the generator was provided with a special filter to reduce the effects of 60-Hz ripple and spurious switching transients. The ripple in the output signal from the generator was 0.3% at low power settings, decreasing to 0.05% at full power. An oscilloscope check showed spikes at the 0 crossing point, the amplitude of which was less than the peak value of the ripple voltage. The rise time of the spikes was 5μs and the decay 15μs. During

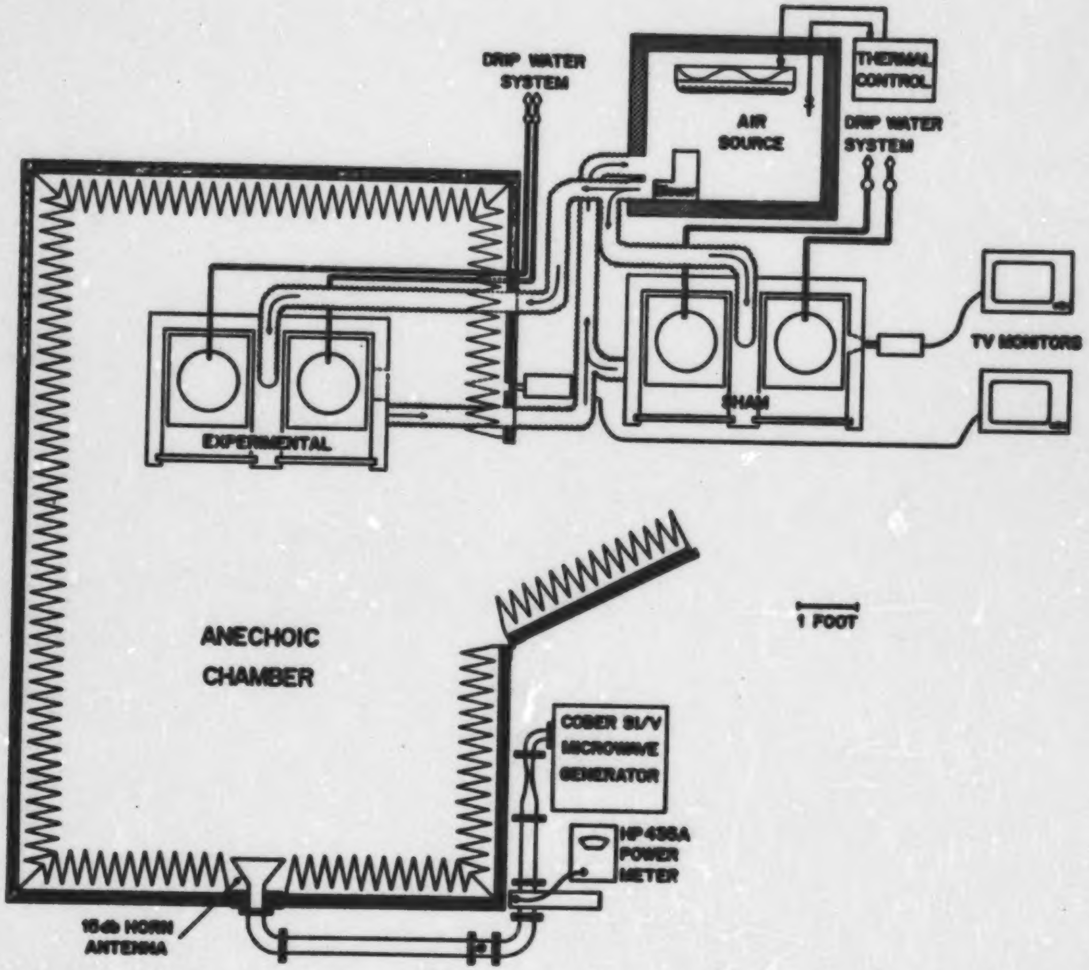
DRIP WATER SYSTEM
THERMAL CONTROL
AIR SOURCE
DRIP WATER SYSTEM
TV MONITORS
SHAM
EXPERIMENTAL
ANECHOIC CHAMBER
1 FOOT
COBER 51/V MICROWAVE GENERATOR
HP 435A POWER METER
15db HORN ANTENNA

Figure 1. Schematic diagram, as viewed from above, of the complete system for microwave and sham exposure of pairs of squirrel monkeys at controlled environmental temperature. Arrows show direction of climate-conditioned air through the 2 exposure chambers. See text for details.

operation, forward power from the generator was monitored by a Hewlett Packard Model 8481A Power Sensor and a Hewlett Parkard Model 435A Power Meter.

b. Exposure Chambers and Cages

Two 61 x 91 x 122 cm exposure chambers, constructed of 5 cm-thick HD-300 Styrofoam (Dow-Corning) housed pairs of cylindrical Plexiglas exposure cages. Each chamber featured a perforated false ceiling which directed climate-conditioned air through the top of each cage, a single exit port for air-return, and a pair of vertically-sliding doors to allow introduction of the cages. One Styrofoam chamber was located in the far field inside the anechoic chamber (microwave-exposed animals); the other Styrofoam chamber (sham-exposed animals) was located on a support structure immediately outside the shielded room (see Figure 1). Special fittings constructed of Styrofoam allowed both chambers to be connected to a single air-conditioning source via 4" diameter Gortiflex tubing (A&A Mfg. Co., New Berlin, WI). This source included a metered flow of fresh air and was capable of producing any ambient temperature from 10 to 40 °C (±1 °C). Relative humidity of the conditioned air was 45 ± 5% and the air flow through the chambers was 235 m/min. Internal illumination of both chambers was provided by fiberoptic systems (Dolan Jenner Industries, Woburn, MA).

Individual exposure cages consisted of a 30.5 cm diameter Plexiglas cylinder, 61 cm high with a stationary ceiling and sliding floor made from 1.3 cm polycube louvers. The cylinder rested on Plexiglas support rods within a shallow polyethylene tray. A single Plexiglas rod, located inside the cylinder 10 cm above the floor, provided a perch for the animal. To minimize focussing of microwaves, the cylinder thickness was

3.1 mm for the microwave-exposed animals, whereas it was a less-fragile 6.3 mm for the sham-exposed. Polished plexiglas tubing cemented at angles in the rear wall of each cylinder permitted drops of water (∿ one/s) to fall after the manner described by Guy (1979). Waste water flowed through Tygon tubing from a capture funnel located behind the cylinder to an open drain in the laboratory.

c. Field Measurements

The far field was mapped at 5-cm intervals across three planes, each orthogonal to the direction of propagation of the incident planewave. These planes were located 1) 245, 2) 255, and 3) 265 cm from the leading edge of the antenna and were 1) tangent to the front of the Plexiglas cylinders, 2) the vertical bisector of the cylinders, and 3) tangent to the rear of the cylinders. Forward power was 500 W. These measurements were made by personnel of the Environmental Protection Agency using EPA field probe #3. A contour plot of the central plane, 255 cm from the antenna, is shown in Figure 2. Superimposed on the plot are the outlines of the two cylindrical cages occupied by the monkeys during chronic microwave exposure. The measurements showed that the central portion of the far field encompassing the cages exhibited a maximum nonuniformity of 8% in the absence of the animals.

d. Dosimetry

Dosimetric determinations were of two types. An approximation of whole-body microwave energy absorption at a power density of 15 mW/cm^2 was based on temperature increments produced by 10-min exposures in a 0.75-liter cylindrical Styrofoam model filled with physiological saline.

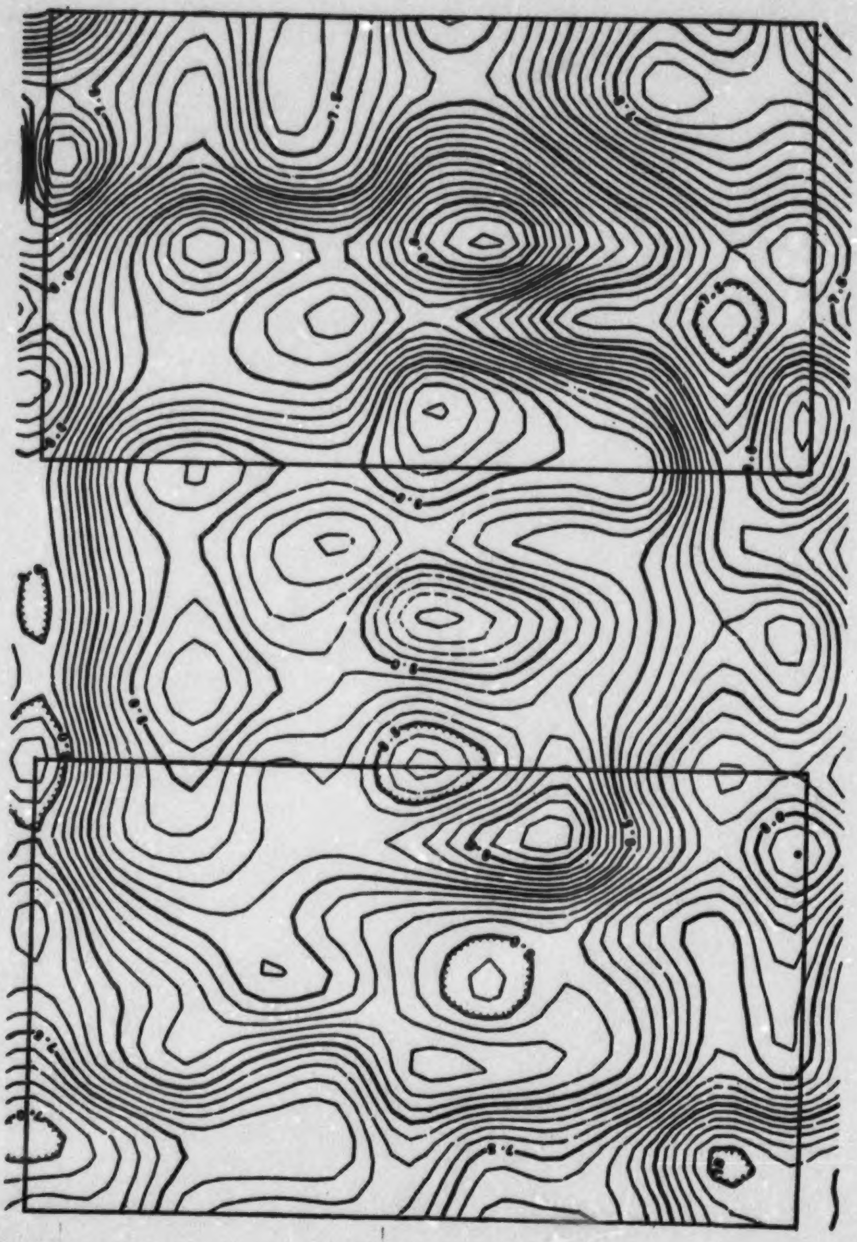

Figure 2. Contour plot of field strength across a 60x80 cm plane, orthogonal to the K vector, centered on the antenna boresight 2.55 m from the leading edge of the antenna. Rectangular outlines show location of two cylindrical Plexiglas exposure cages in the field, absent during the measurements. Forward power=500 W.

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The interior dimensions of the model, 5.2 cm in diameter x 33 cm high, were an idealized approximation of a standing squirrel monkey. Temperatures were measured at four depths within the liquid as described by Adair and Adams (1980b) when the model was positioned in the center and at the four primary compass points inside one Plexiglas exposure cylinder. For these measurements, the cylinder was positioned on the antenna boresight at a mean distance of 255 cm from the antenna aperture. The mean temperature rise above an equilibrated 25 °C was used to calculate the specific absorption rate (SAR): SAR = 0.16 (W/kg)/(mW/cm^2), a value comparable to that predicted by Durney, *et al.* (1978). Power densities used for chronic exposure in the experiments reported here were 1 and 5 mW/cm^2, representing whole-body SARs of 0.16 and 0.8 W/kg.

Additional dosimetric procedures were conducted by personnel of the Environmental Protection Agency (Kinn, 1977). A Styrofoam model of a perched monkey was filled with 706 g of tissue-equivalent material (Guy, 1971). The model was placed inside one of the cylindrical Plexiglas cages, at its normal location in the far field, and exposed to the microwave field at 18 mW/cm^2 for 10 min. The contents of the model was removed quickly and placed in one well of a twin-well calorimeter. An equal mass of material, implanted with a heating element, was placed in the opposite well. Using the calorimeter as a "thermal balance", heat was added to the unexposed material and the summed power applied to the heater after thermal equilibrium was reached was used to compute whole-body SAR. Four trials for a frontal exposure of the model yielded an average SAR of 0.21 (W/kg)/(mW/cm^2) and for a lateral exposure an average SAR of 0.16 (W/kg)/(mW/cm^2).

3. Physiological Tests

Effect(s) of chronic temperature and/or microwave exposure were assessed by determining thermoregulatory responses of individual animals to a series of ambient temperature challenges. Standardized tests were conducted on each animal prior to chronic exposure, to establish reliable baseline levels of response, and at predetermined times during and after chronic exposure. Measurements of skin temperatures, colonic temperature (T_{co}) and oxygen consumption were made during the tests. In addition, water content of air circulated over the plantar surface of the foot was monitored to determine both onset and rate of sweat production.

a. Physiological Test System

Figure 3 shows the basic elements in the physiological test system (not drawn to scale). All tests were conducted in an insulated, aluminum-clad environmental chamber of interior dimensions 92 x 102 x 66 cm. Air flowed through the chamber from an outside source at a constant rate of 20 liters/min. Air temperature (T_a) in the chamber was controlled at specific fixed temperatures (±0.5 °C) using a proportional temperature controller. At the beginning of each test, the monkey was restrained in a Plexiglas chair and thermocouples attached for measurements of colonic and skin temperatures. Copper-constantan thermocouples (36-ga) in special configurations were used with reference junctions located in a bath of melting ice and water. T_{co} was determined using a lubricated, polyethylene-encased thermocouple inserted 10 cm. beyond the anal sphincter. Skin temperature measurements were made from the plantar surface of the left foot and 3 shaved skin areas, the right calf,

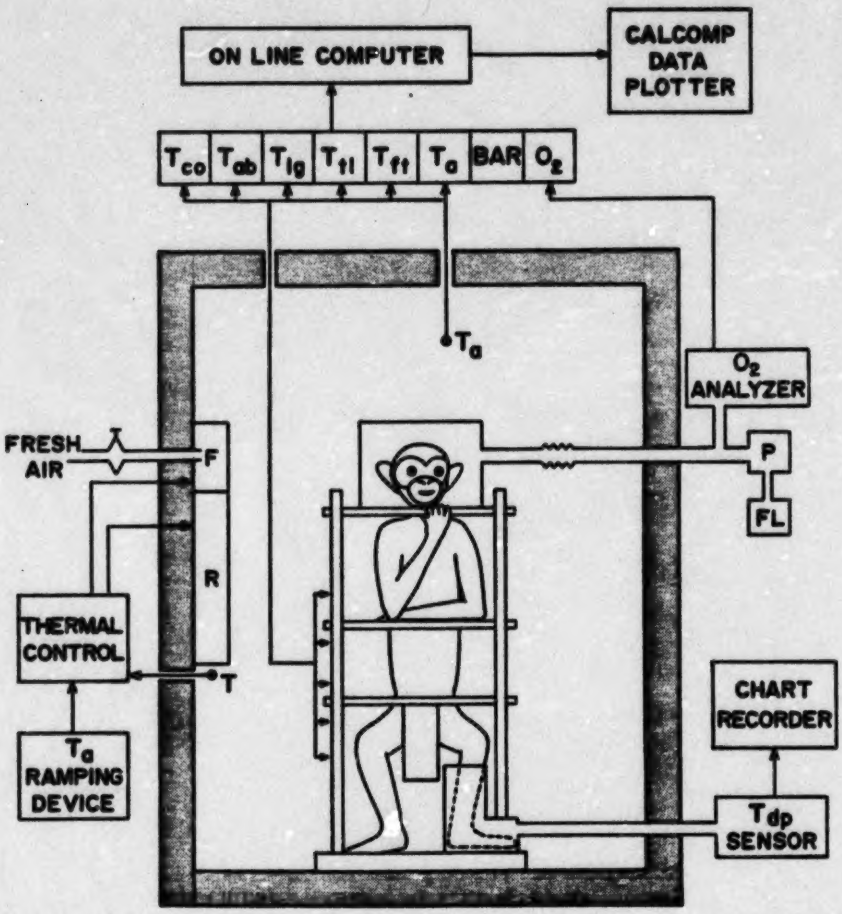
ON LINE COMPUTER
CALCOMP DATA PLOTTER
T_{co}
T_{ab}
T_{lg}
T_{tl}
T_{ft}
T_a
BAR
O_2
T_a
O_2 ANALYZER
FRESH AIR
F
P
FL
R
THERMAL CONTROL
T
T_a RAMPING DEVICE
CHART RECORDER
T_{dp} SENSOR

Figure 3. Schematic Diagram of the components of the physiological test apparatus and response measures (not drawn to scale). Ambient temperature of test chamber (T_a), sensed by thermistor (T) is controlled by fans (F) and heat exchanger (R). Monkey's expired air is drawn from a hood by a pump (P) at a constant flow (FL) for analysis of oxygen deficit (O_2) and calculation of metabolic heat production. Foot sweating is assessed by changes in dewpoint temperature (T_{dp}) of air passing over the foot. Barometric pressure (BAR), colonic (T_{co}) and skin temperatures (T_{ab}, T_{lg}, T_{tl}, T_{ft}) are monitored every minute by an on-line computer that displays reduced data on a Calcomp plotter.

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abdomen, and dorsal surface of the base of the tail. A weighted value of mean skin temperature was calculated from these skin temperature measurements according to Stitt, et al. (1971):

$$\bar{T}_{sk} = 0.07\ T_{foot} + 0.37\ T_{leg} + 0.45\ T_{abdomen} + 0.11\ T_{tail}.$$

Oxygen consumption was measured using an open-flow system. A Plexiglas hood was placed over the head of the animal and chamber air was drawn through the hood at a constant flow rate of 7.3 l/min. Oxygen content of a portion of the effluent air was measured, using a Beckman Model 755 paramagnetic oxygen analyzer, and metabolic heat production (M) calculated assuming constant RQ value of 0.83.

All body temperatures and oxygen consumption were sampled at 1-min intervals by both central and peripheral data acquisition systems and displayed on a Calcomp Model 565 X-Y plotter. These responses were closely monitored during the tests to ensure minimal stress to the animal.

Sweat rate was measured from the sole of the foot. An L-shaped acrylic boot enclosed the right foot of the animal. The sole rested on a mesh screen support inside the boot. Air flowed over the sole at a constant flow rate of 1.9 l/min. Water vapor content of the effluent air was determined using a dew point temperature sensor located outside the environmental chamber (Wood, 1970). Output of the dew point monitoring circuit was from a thermocouple whose voltage was directly proportional to the dew point temperature (Graichen, et al., 1982). The dew point temperature of boot effluent air was monitored constantly and displayed on a strip chart.

b. Test Protocol

Each animal was placed in the chamber equilibrated at T_a = 20 °C ± 0.5 °C and allowed a minimum of 1 h to adjust to the environmental conditions. After all measured variables had reached steady-state levels, T_a was increased in ramp form to 26.6 ± 0.5 °C and maintained at this level for approximately 45 min. After exposure to 26.6 °C, T_a was incremented automatically in ramps of 3.3 ± 0.5 °C at approximate intervals of 37.5 min, yielding test T_a of 29.9, 33.2 and 36.5 °C. Exposures above 20 °C were transient in nature, never extending for the length of time required to establish steady-state responses. Abbreviated exposures of this type were required to minimize the test time and the possibility of stress due to restraint. A typical printout of data collected during a single experiment is shown for one monkey (S:Pippin) in Figure 4. The mean of the last five samples of each variable at the end of each T_a exposure period (hatched bars) was calculated and used for all comparisons.

Figure 5 shows a record of water loss from the sole of the right foot of one monkey (S:Pippin) collected during a physiological test. In general, no increase in evaporation directly attributable to sweat activity occurred until T_a=36.6 °C. If active sweating was not evident at the end of exposure to this T_a, the temperature was slowly increased to initiate the response. Each monkey was removed from the chamber when T_{co} approached 40.0 °C to avoid severe heat stress.

Statistical analyses of several of the physiological variables were accomplished using a two-way ANOVA set at $p \leq 0.05$. These analyses were performed to determine both individual and interactive effects of chronic

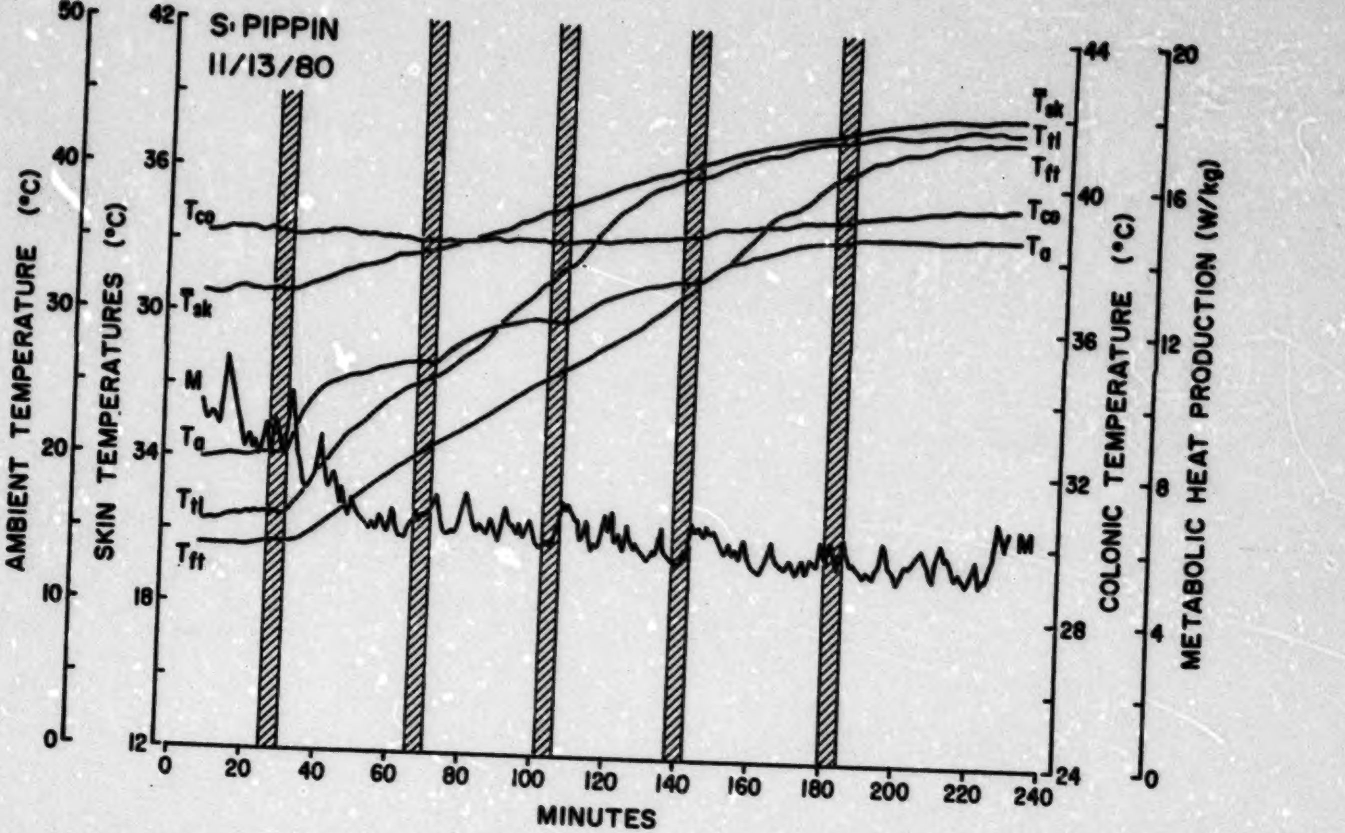

Figure 4. Colonic temperature (T_{co}), mean skin temperature ($\bar{T}_{sk}$), metabolic rate (M), chamber air temperature (T_a), tail skin temperature (T_{tl}) and foot skin temperature (T_{ft}) as a function of time during a standard physiological test. Hatched vertical bars indicate the 5 min. period at the end of exposure to each test T_a when mean value of each variable was calculated.

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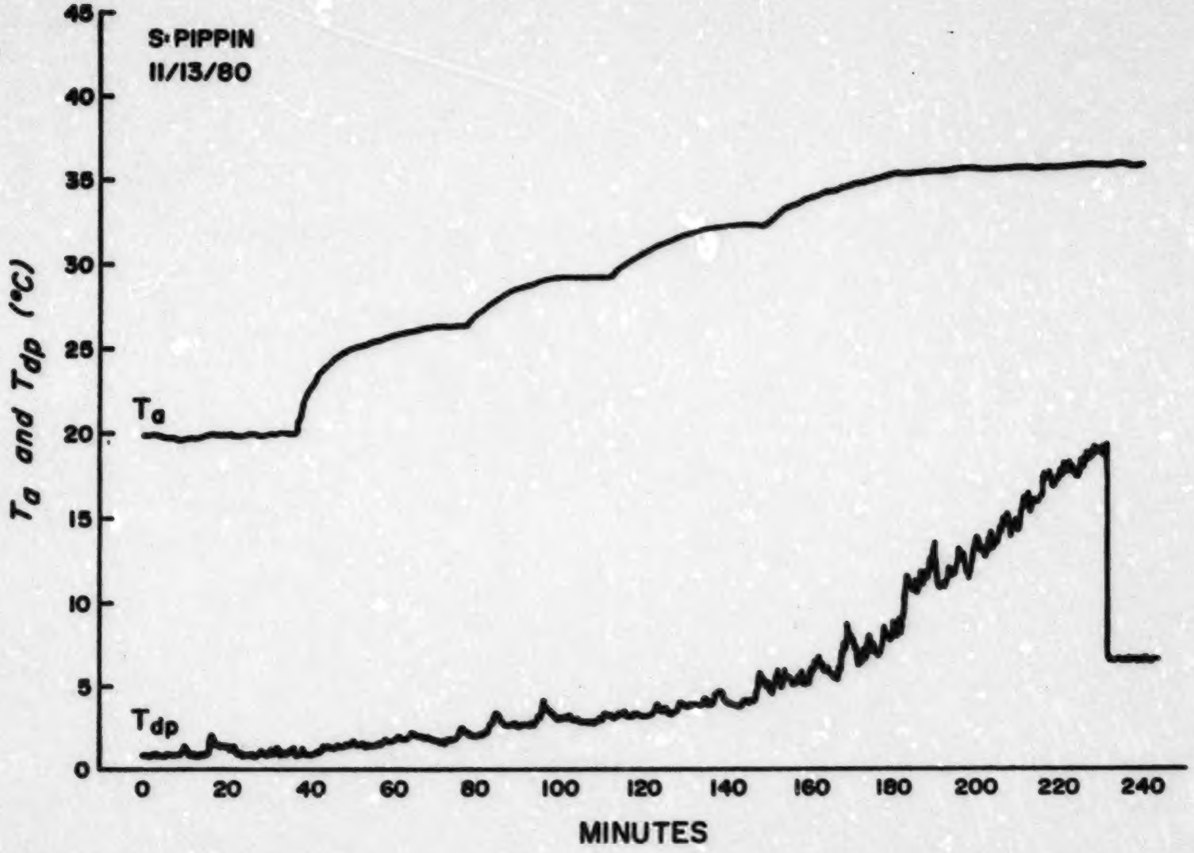

Figure 5. Chamber air temperature (T_a) and dewpoint temperature (T_{dp}) of "boot" air measured during a physiological test.

microwave and/or T_a exposure on thermoregulatory responses. More detailed statistical analyses to determine specific effects were not possible due to limitations in sample size of each group (i.e., n=2).

4. Behavioral Tests

a. Behavioral Test Chamber

During the behavioral tests, a monkey was chair-restrained in the far field of a 15-dB standard gain horn antenna inside an electromagnetically-anechoic chamber of interior dimensions 1.8 x 1.8 x 2.5 m. The monkey was enclosed by a 30 x 51 x 33 cm Styrofoam box through which a temperature-controlled airstream was directed, as shown in Figure 6. A valve system allowed air from one of two regulated (±0.5 °C) sources to circulate at 0.36 m/s through the box. The monkey was trained to pull a response cord to operate the valves, thereby selecting between two preset air temperatures, 10 and 50 °C. During the tests, the monkey was exposed to 10 °C air and each response was reinforced by a 15-sec presentation of 50 °C air. The 10 °C air then automatically returned until the animal responded again. Over a period of several hours, highly-trained monkeys usually select an average air temperature of 35-36 °C. Selected air temperature (T_a selected) was measured by a copper-constantan thermocouple in the air outlet from the anechoic chamber and was recorded continuously on a strip chart. The temperature tracing also provided a temporal record of the animal's behavioral responses.

During all behavioral tests, the following body temperatures were read once a minute by an on-line computer: colonic temperature (T_{co}) and four representative skin temperatures (T_{sk}) measured on the abdomen,

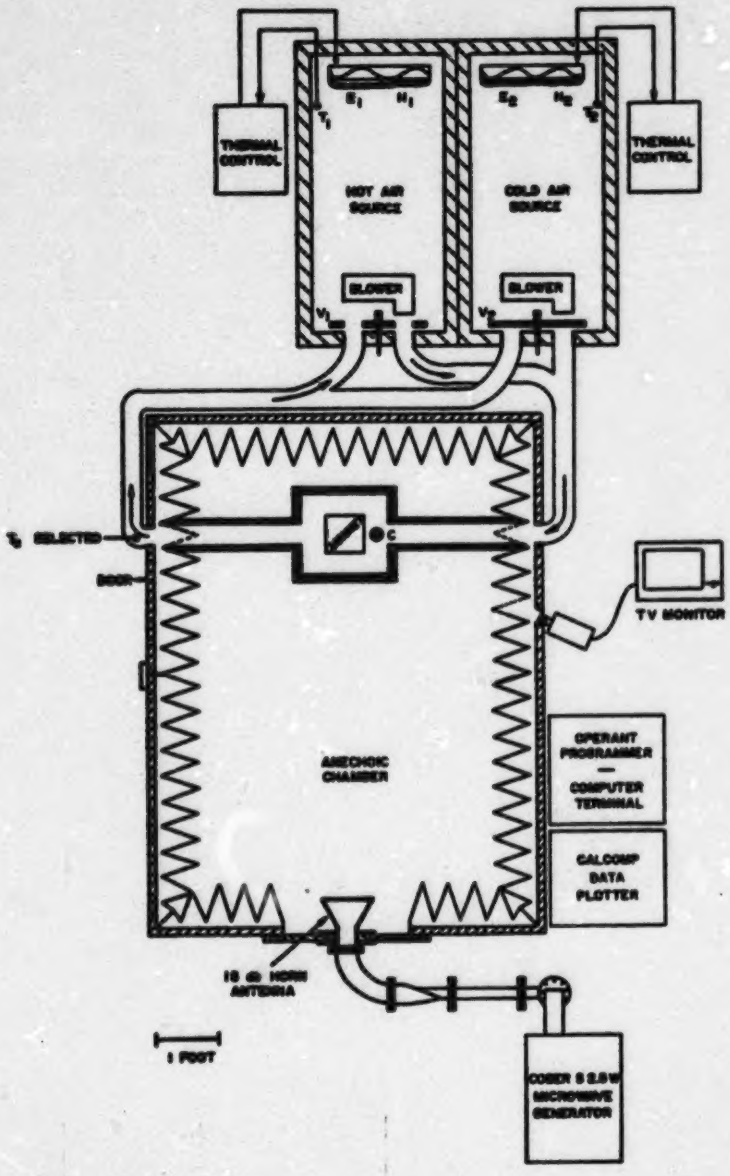
THERMAL CONTROL
T_1
E_1
H_1
HOT AIR SOURCE
BLOWER
V_1
THERMAL CONTROL
E_2
H_2
T_2
COLD AIR SOURCE
BLOWER
V_2
T_a SELECTED
DOOR
C
TV MONITOR
ANECHOIC CHAMBER
OPERANT PROGRAMMER
—
COMPUTER TERMINAL
CALCOMP DATA PLOTTER
18 db HORN ANTENNA
1 FOOT
COBER S 2.5 W MICROWAVE GENERATOR

Figure 6. Schematic diagram (as viewed from above) of anechoic chamber, microwave generator and transmission system, basis elements in the air control system for tests of behavioral thermoregulation. Valves (V_1 and V_2) operated by the monkey's response cord (c) determine air flow through Styrofoam box enclosing monkey. Air source temperatures, sensed by thermistors (T_1 and T_2) are controlled by heat exchangers (E_1 and E_2) and coil heaters (H_1 and H_2).

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tail, leg, and foot (cf. Adair and Adams, 1980b for details). From the four skin temperatures, a weighted mean skin temperature was calculated as described above.

The monkey was under constant video surveillance during the tests. These were conducted in the presence of a 73-dB (SPL) masking noise to prevent auditory cues to the presence or absence of microwaves (Green, 1975).

Continuous microwaves at a frequency of 2450 ± 25-MHz were generated by a 2.5-kW source (Cober Electronics Model S2/W) and fed to the antenna through standard waveguide. The electric field vector was aligned with the long axis of the monkey's body (E-polarization). Forward power was monitored with a Heath digital power meter. Calibration measurements of the far field across a 1.0 x 1.5 m plane that passed through the restraining chair location orthogonal to the K vector of the incident planewave are described in detail by Adair and Adams (1980b). The maximal nonuniformity of the central 50 x 50 cm of the plane (encompassing the monkey's location) was 8% with the chair absent and 13% with the chair present. Power densities used in the behavioral tests were determined with a Narda Model 9323 field probe positioned, with chair present, at the location of the monkey's head.

An approximation of whole-body absorption of microwave energy over a range of power densities from 5-40 mW/cm^2 was based on temperature increments produced by 10-min exposures of a 1.1 liter cylindrical Styrofoam model filled with physiological saline. Details of this procedure appear in Adair and Adams (1980b). The whole-body specific absorption rate (SAR) determined by this method was 0.15 $(W/kg)/(mW/cm^2)$.

The power densities used in the behavioral tests ranged from 4 to 12 mW/cm^2, representing whole-body SARs from 0.6 to 1.8 W/kg.

b. Behavioral Test Protocol

Following extensive training in the behavioral response, each experimental animal underwent a minimum of three 4-h baseline test sessions of behavioral thermoregulation. These established the normally-preferred T_a and pattern of responding as well as the skin and colonic temperatures achieved thereby. Then, prior to the initiation of chronic microwave or sham exposure, each animal was given three standardized behavioral tests that were designed to determine the microwave intensity (whole-body exposure) that would reliably alter thermoregulatory behavior. Modeled after the protocol described by Adair and Adams (1980b), these tests began with a 90-min baseline period during which no microwaves were present. The baseline was followed by five 10-min exposures to 2450-MHz CW microwaves at increasing power density (4,6,8,10 and 12 mW/cm^2) separated by periods of like duration when no microwaves were present.

To illustrate the behavioral protocol, Figure 7 shows representative data from the behavioral tests conducted on one monkey prior to chronic microwave exposure. The plotted points represent mean values calculated over the preceding 10-min period across three tests spaced approximately one week apart. Open symbols represent data collected during three baseline tests during which no microwaves were present. The error bars on the functions for T_a selected indicate 1 SEM (based on 30 values). The threshold for microwave alteration of thermoregulatory behavior was

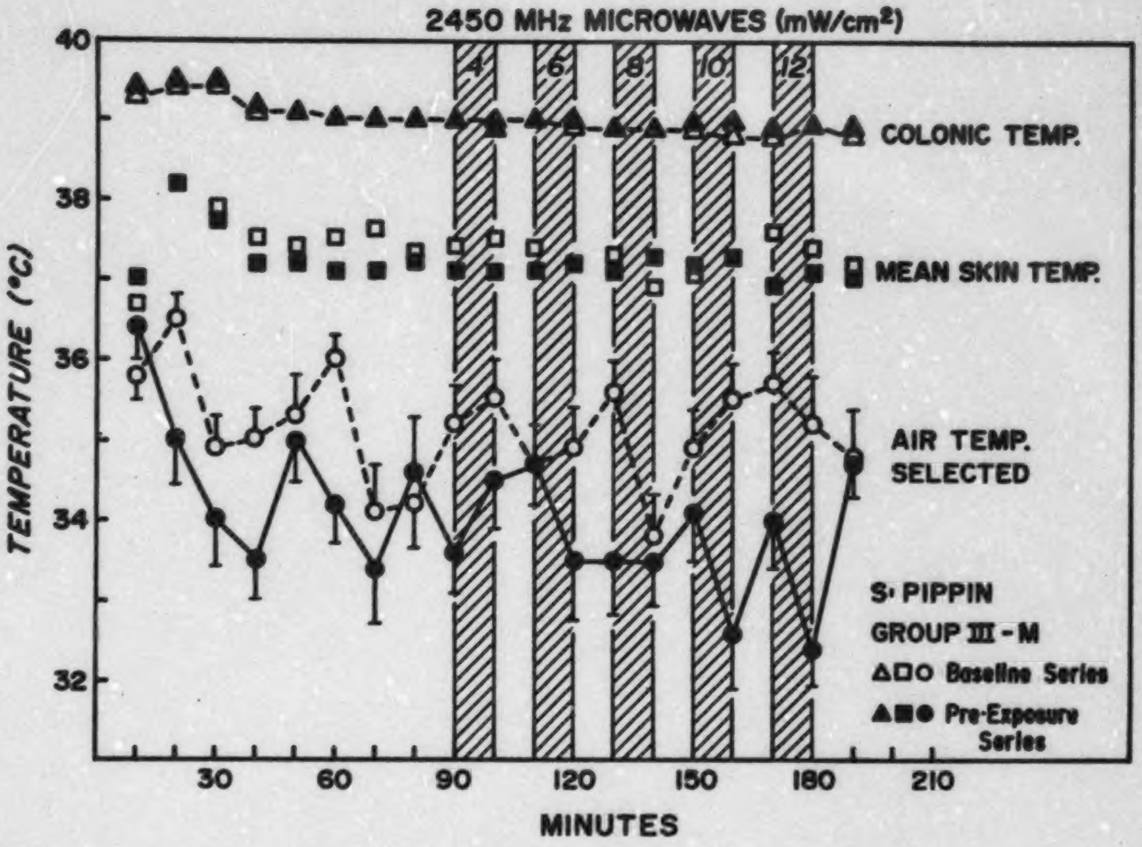

Figure 7. Mean colonic temperature, weighted mean skin temperature, and air temperature (±1 SEM) selected by one monkey as a function of experimental time, to illustrate the protocol for the tests of behavioral termoregulation. Open symbols represent mean data from 3 baseline tests during which no microwaves were present. Solid symbols represent mean data from 3 tests prior to chronic exposure that involved five 10-min exposures to 2450-MHz CW microwaves at increasing power density (4,6,8,10 and 12 mW/cm^2).

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defined as that power density at and above which there was a significant difference in T_a selected between the baseline and microwave tests. In practice, a significant difference was inferred from non-overlapping standard errors. Thus, for the data presented in Figure 7, 10 mW/cm^2 is the microwave threshold for the alteration of thermoregulatory behavior. While the two points at 6 mW/cm^2 are, by our criteria, significantly different, those at 8 mW/cm^2 are not.

Behavioral tests, using this protocol, were conducted every four weeks during and after chronic microwave (or sham) exposure (see Experimental Design below). It should be noted that the test protocol for Group I animals was slightly different from that described above, in that a shorter (60-min) baseline period preceded the microwave exposures which were usually four, rather than five in number.

5. Blood Tests - (Groups I-III)

A total of 5 ml of blood was withdrawn via femoral venipuncture from unanesthetized animals for analysis. All samples were taken at the weigh-in on Wednesday morning of weeks -1,5,10,15, and 20 of the treatment period (see Figure 9 below). No physiological or behavioral test was conducted on any day which coincided with a blood-sampling day, to minimize stress to the animal. Of the 5-ml sample, 2 ml were immediately placed in an EDTA tube for whole blood analyses and the remainder used for serum analyses. All cell counts and hemoglobin determinations were performed using standard hematology procedures.

Measurements of total serum thyroxine concentration and thyroxine binding capacity were performed using competitive protein binding techniques. Blood sodium, potassium, bicarbonate and chloride concentrations were determined by flame emission. Total serum protein and albumin concentrations were determined using the biuret reaction and bromcresol green binding methods, respectively.

6. Experimental Design and Test Sequence

As stated above, this research project was designed to identify and measure changes in thermoregulatory response systems, physiological and behavioral, that may occur when squirrel monkeys are exposed to 2450-MHz CW microwaves at controlled environmental temperatures 40 h/wk for 15 weeks. Two microwave power densities were explored, 1 and 5 mW/cm^2, and three ambient temperatures, 25, 30, and 35 °C, making a total of six treatment groups. The groups were numbered (I-VI) in accordance with the order in which they were run. Figure 8 diagrams the basic 2x3-cell experimental design. Each cell of the matrix contained 4 animals, two microwave-exposed (designated M in the inset) and two sham-exposed, (designated S). Thus, an animal designated as group V-M was chronically exposed to microwaves at a power density of 5 mW/cm^2 and an ambient temperature of 30 °C.

The temporal treatment and sequence of tests was the same for all groups and is diagrammed in Figure 9. The temporal protocol was divided into three phases as follows:

1) Pre-exposure phase of 8-12 weeks duration. During this period, each animal was given four standard tests of physiological thermoregulation which constituted baseline data. Each animal was given at least three baseline tests of behavioral thermoregulation (without microwave

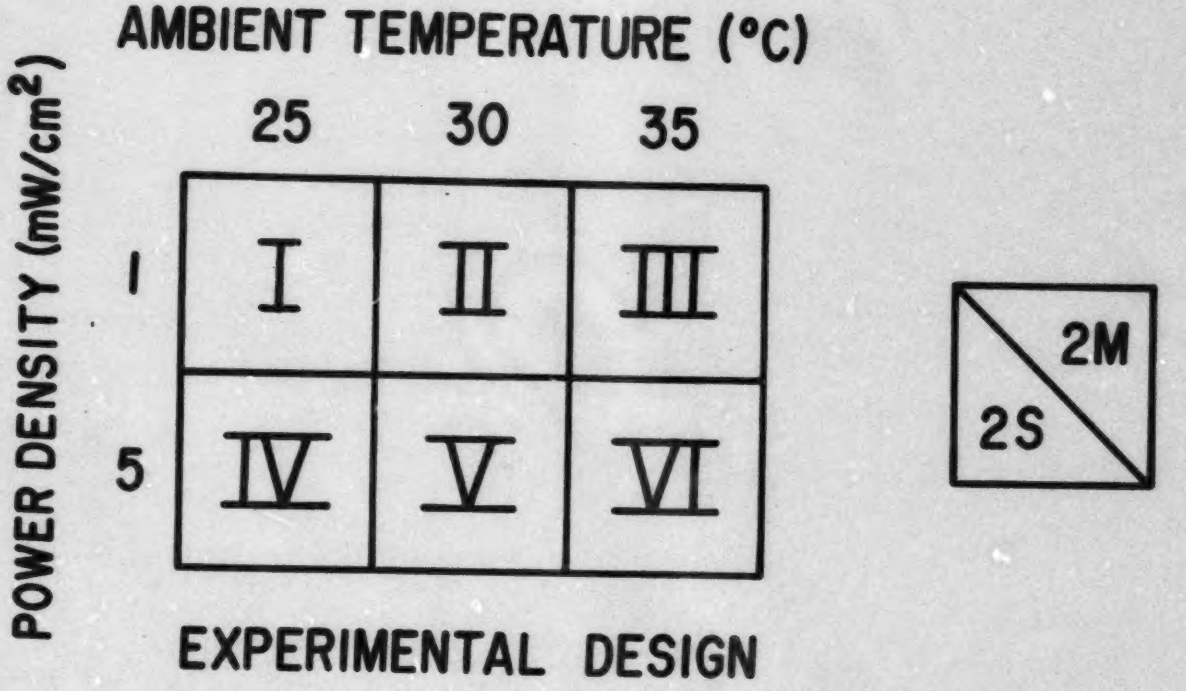
AMBIENT TEMPERATURE (°C)
25
30
35
POWER DENSITY (mW/cm²)
1
5
I
II
III
IV
V
VI
2M
2S
EXPERIMENTAL DESIGN

Figure 8. Design matrix for the research project indicating, by Roman numerals, the numbers of individual groups. Each cell of the matrix contains four animals, two microwave-exposed (M) and two sham-exposed (S).

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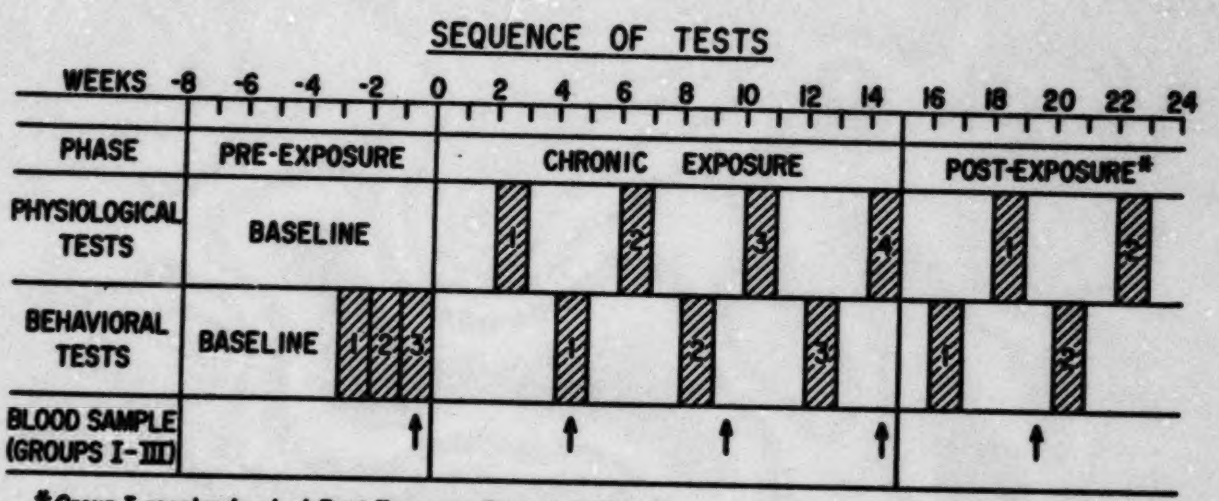
SEQUENCE OF TESTS
WEEKS
-8
-6
-4
-2
0
2
4
6
8
10
12
14
16
18
20
22
24
PHASE
PRE-EXPOSURE
CHRONIC EXPOSURE
POST-EXPOSURE*
PHYSIOLOGICAL TESTS
BASELINE
1
2
3
4
1
2
BEHAVIORAL TESTS
BASELINE
1
2
3
1
2
3
1
2
BLOOD SAMPLE (GROUPS I-III)

*Group I received only 1 Post-Exposure Test of each type

Figure 9. Temporal protocol and sequence of tests for each group.

exposure) and three standard behavioral tests to determine the microwave threshold to alter thermoregulatory behavior. During this period, each animal spent one or two days/wk in a Plexiglas cylindrical cage in the laboratory environment. A 5 ml venous blood sample was collected during the last week before chronic exposure was initiated (Groups I-III). Animals were weighed weekly.

2) Chronic microwave- or sham-exposure phase of 15 weeks duration. Ambient temperature was constant (e.g. 30 °C) as was power density (e.g. 1 mW/cw^2) throughout the exposure period. The two microwave-exposed monkeys occupied the Plexiglas cylinders in the Styrofoam enclosure within the anechoic chamber from 0830 to 1630 hrs Monday through Friday; the two sham-exposed monkeys occupied the Plexiglas cylinders in the sham-exposure chamber simultaneously. During this period, each animal was given four standard physiological tests and three standard behavioral tests in the weeks indicated in Figure 9. A blood sample was collected during weeks 5,10, and 15 (Groups I-III). Animals were weighed weekly.

3) Post-exposure follow-up phase of 4-8 weeks duration. The procedures followed were similar to those in the first 8 weeks of the chronic exposure period and consisted of two standard physiological tests and two standard behavioral tests. (Note that Group I animals received a single post-exposure follow-up test of each type.) One blood sample was taken during this period. Animals were weighed weekly.

RESULTS

a. Body Mass

Each week, the measurement of body mass for each animal was entered on a graph that spanned all phases of the animal's participation in a given group, a period approximating 6 months. The area under this curve was determined by integration for each phase of treatment (i.e. pre-exposure, chronic exposure, post-exposure) so as to yield a measure of average body mass for a given treatment condition. From these measures, changes in body mass from one phase to another could also be determined.

The data were first examined for evidence of seasonal variation in body mass, a potential source of variability in feral-born animal subjects of differing ages. Half of the groups (I, III, and V) were run during a period from summer (April-June) to winter (November-January); the other half (Groups II, IV, and VI) were run from winter to summer. The body masses of all the animals in each half, determined across each phase of treatment, were averaged irrespective of the specific conditions under which each animal was chronically exposed. Figure 10 shows the mean body mass (±1 SEM) for each half of the total subject population across the three phases. Although there was a tendency for body mass to increase from the chronic-exposure phase to the post-exposure phase in the summer-winter half of the population, there was no statistically significant difference between the two sub-populations at any given treatment phase.

The change in body mass that may be attributable to effects of chronic exposure is represented by the difference between pre-exposure and chronic exposure means. This measure is presented for each group in Figure 11. The data for sham-exposed animals (n=4) are pooled from the

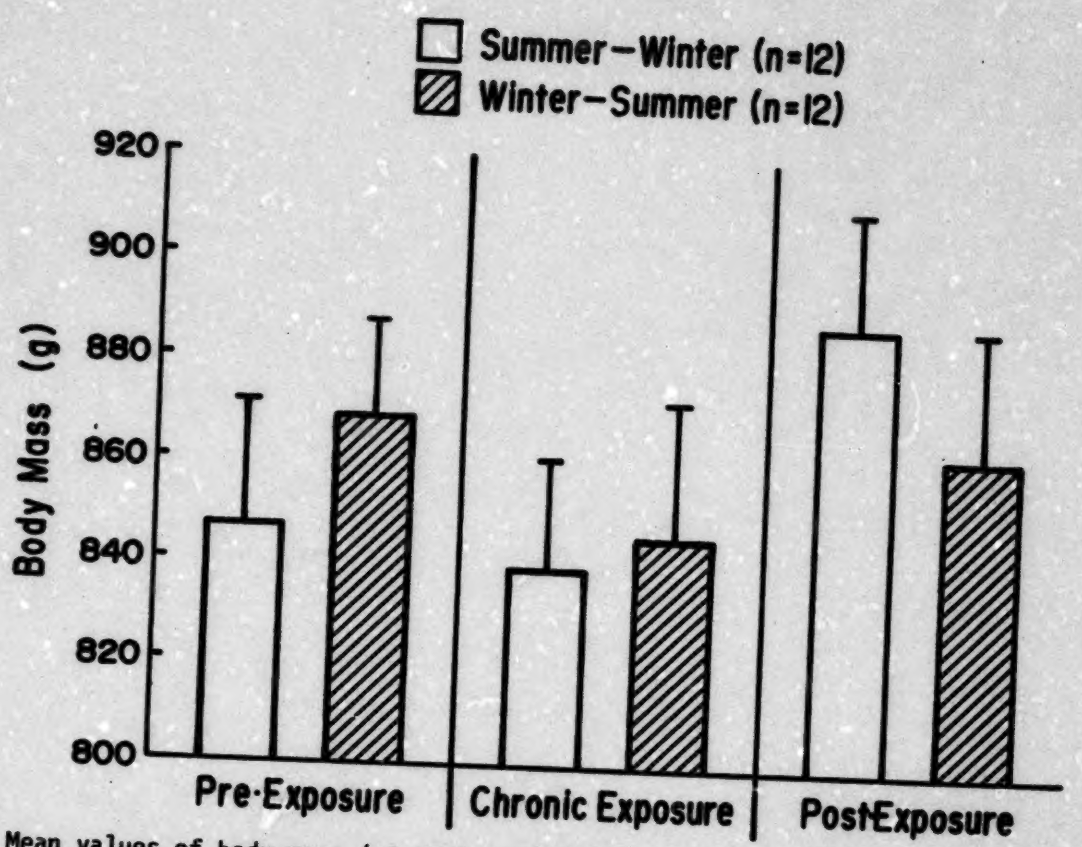

Figure 10. Mean values of body mass (±1 SEM) determined for each animal by integration over the entire duration of each phase and separated into groups on the basis of seasonal exposure.

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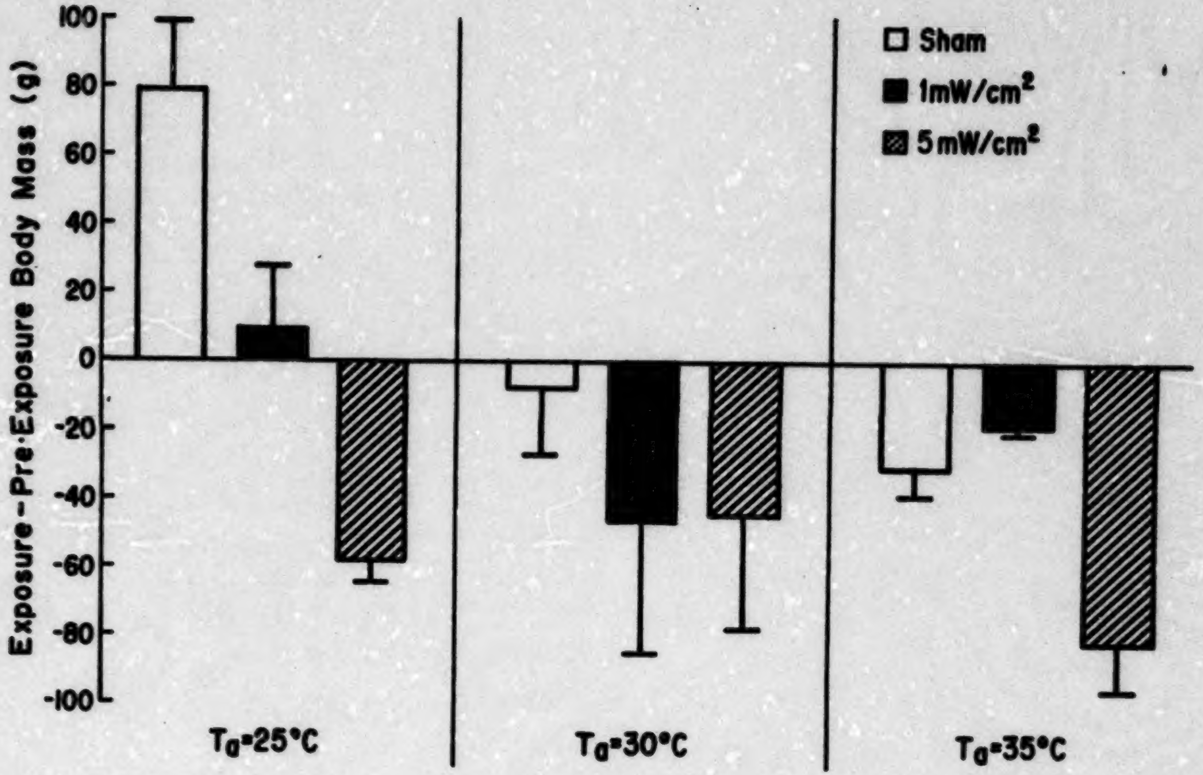

Figure 11. Differences in mean values of exposure and pre-exposure body mass ($\underline{+}$1 SEM) determined for each animal by integration of responses over the entire duration of each phase and separated into groups on the basis of chronic exposure treatments. Sample size of each group, regardless of T_a, was 4, 2 and 2 for sham, 1 and 5 mW/cm^2 exposed animals, respectively.

two groups exposed at each T_a. In most cases, body mass changed little or decreased during the chronic-exposure phase relative to the pre-exposure level. However, there was a significant effect of both T_a and microwave exposure on the change in body mass. Sham-exposed animals increased body mass when chronic exposure was conducted at T_a=25 °C but showed no change or a slight reduction of body mass in the warmer environments. Further, although no statistical test for a specific effect was performed, it is apparent from Figure 11 that sham-exposed animals at T_a= 25 °C increased body mass during chronic exposure while 5 mW/cm^2-exposed animals at the same T_a decreased body mass. No significant differences in body mass between these two groups (819.9 ± 50.6 g and 869.1 ±19.0 g respectively) existed during the pre-exposure phase. In addition, all animals exposed at 5 mW/cm^2, irrespective of T_a, experienced an individual decrease in body mass during the chronic exposure phase. These decrements ranged from 11 to 95 g.

b. Physiological Tests

1) Body temperature and metabolic rate

The physiological tests were conducted on each animal at predetermined intervals during the study to assess the thermoregulatory responses of each animal to test T_a of 20.0, 26.6, 29.9, 33.3, and 36.6 °C. All measurements described earlier were made on the animals from all groups, unless stated otherwise. For the pre-exposure (baseline) phase, the results of 4 separate tests on each animal were averaged to derive a mean value representative of the baseline response for the particular individual. An example of such baseline data (body temperatures and metabolic rate) is depicted by solid lines in Figure 12. For the chronic

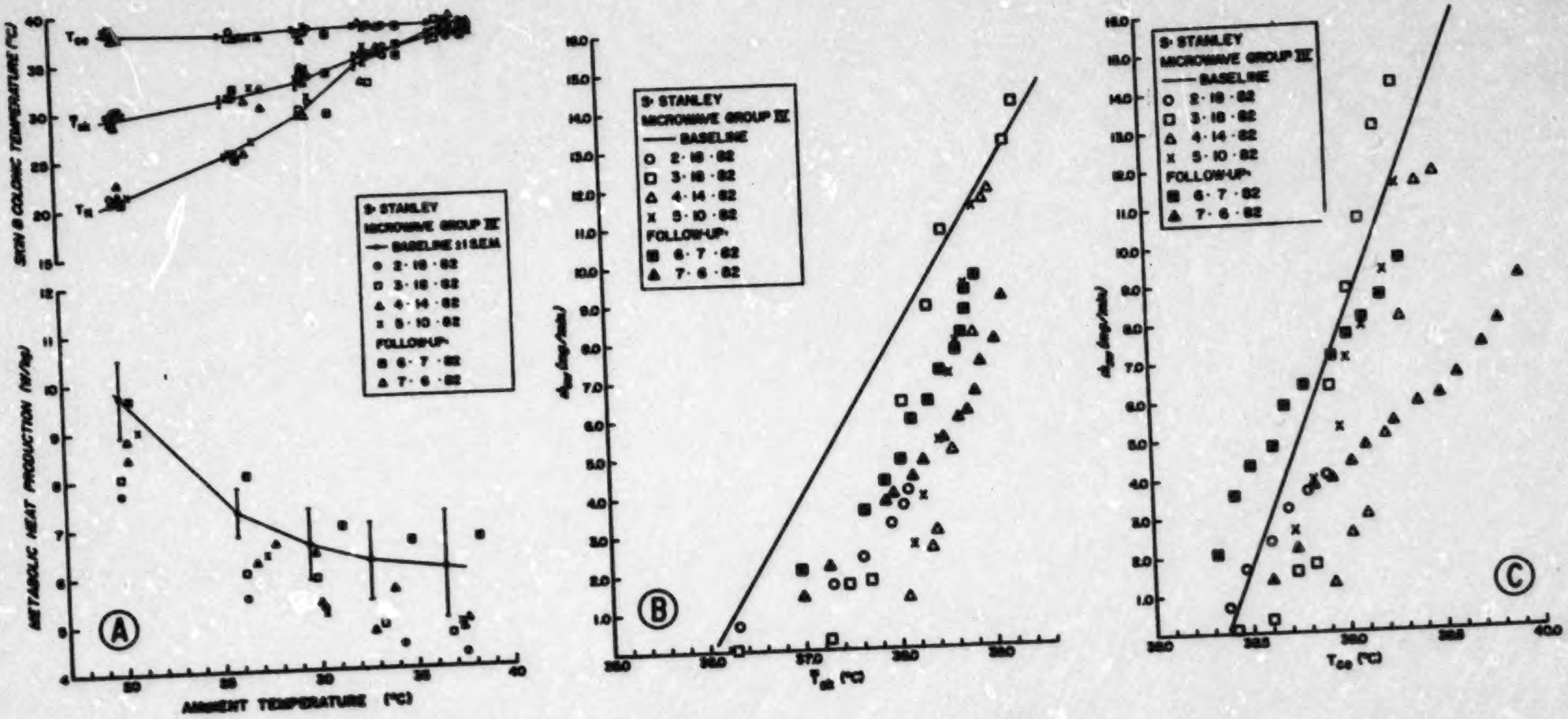

Figure 12. Mean baseline data (——) and data collected from single physiological tests conducted during and after chronic exposure of one animal. Panel A presents colonic temperature (T_{co}), mean skin temperature ($\bar{T}_{sk}$), tail skin temperature (T_{tl}) and metabolic heat production as a function of chamber air temperature. Panel B presents sweat rate ($\dot{m}_{sw}$) as a function of $\bar{T}_{sk}$ and panel C presents the relationship of $\dot{m}_{sw}$ to T_{co}.

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exposure and post-exposure phases, results of individual tests on each animal were compared with the mean baseline values to assess changes at specific points in time. Statistical analyses were performed only on data collected in single tests during the exposure treatment, with no comparisons of data between tests (e.g., test 2 _vs_ test 4). In addition, analyses were performed only on data collected at single test T_a, with no comparison of responses at different temperatures (e.g., test T_a=20.0 °C _vs_ test T_a=29.9 °C).

Differences between groups during the pre-exposure phase were determined using a two-way ANOVA. Although animals had not been chronically exposed or sham exposed to microwaves at a particular T_a, the data were analyzed as if treatment had already occurred to detect possible differences in pre-exposure values. No differences were noted in values of T_{co} and M for Groups I-VI. However, significant differences were found in $\overline{T}_{sk}$, attributed to both a large variability in normal values for this particular dependent variable and the small sample size used.

Responses of M, T_{co}, and $\overline{T}_{sk}$ during tests 2 and 4 conducted during the chronic exposure phase were analyzed for effects of temperature and microwave treatments. Although 4 physiological tests were administered during chronic exposure, the results of only these 2 tests are presented in this report. Potential response changes were predicted to be well under way by test 2, maximal by test 4. Initial comparisons were made using actual values collected during each test. The results of a two-way ANOVA indicated only small effects of the 2 treatment variables on the response variables. For example, chronic microwave effects were noted on responses of T_{co} and $\overline{T}_{sk}$ at test T_a=33.3 and 36.6 °C. Ambient temperature effects on $\overline{T}_{sk}$ and M were found only at test T_a=36.6 °C, and on T_{co} at test T_a=26.6 °C. No interactive effects on $\overline{T}_{sk}$ and M responses were noted. Such effects, albeit significant, should be accepted with extreme caution in view of the small sample size of each group.

Physiological thermoregulatory responses recorded during the exposure and post-exposure phases must be considered in the context of pre-exposure levels to determine if, in fact, a change in response did occur as a result of a particular chronic exposure treatment. Mean values of baseline responses for each animal at each test T_a were subtracted from the responses recorded during tests 2 and 4 of the chronic exposure phase to yield delta values. Variance analysis of the results of test 2 showed no effect of either temperature or microwave treatment on ΔT_{co} or ΔM measured at each of the 5 test temperatures. However, $\Delta\overline{T}_{sk}$ was significantly affected by both temperature and microwave exposures over a range of test T_a from 26.6 to 36.6 °C, with major effects appearing over the test T_a of 29.9 to 36.6 °C. In addition, a significant interaction of temperature and microwave treatments on $\Delta\overline{T}_{sk}$ was noted at the test T_a of 33.3 and 36.6 °C. A similar analysis of the results of test 4 showed no effect of either treatment on ΔT_{co} at any test T_a, with only a slight effect of ambient temperature on ΔM at test T_a=33.3 and 36.6 °C. The ambient temperature prevailing during chronic exposure significantly affected $\Delta\overline{T}_{sk}$, with the largest effect occurring at test T_a=29.9 °C. The effect of microwave exposure on $\overline{T}_{sk}$ values measured during test 4 was only apparent at test T_a=29.9 °C. At the same time, an interactive effect of microwave and temperature treatments on $\overline{T}_{sk}$ was small, occurring only at test T_a=29.9 and 36.6 °C.

It is possible that the observed changes in $\overline{T}_{sk}$ from the baseline level were only a reflection of the significant differences in this variable which were present during the pre-exposure phase. At test T_a=29.9 °C, the $\overline{T}_{sk}$ of animals chronically exposed to an ambient temperature of 25 °C increased from a baseline value of 33.18 ± 0.21 °C to 33.51 ± 0.13 °C during test 2 of the chronic exposure phase, whereas animals exposed to 35.5 °C decreased $\overline{T}_{sk}$ from 33.97±0.21 °C to 33.19±0.20 °C over the same time period. In brief, animals that had a low $\overline{T}_{sk}$ during the pre-exposure phase increased their response with exposure while animals with a high $\overline{T}_{sk}$ showed the reverse trend. Figure 13 displays the mean values of $\overline{T}_{sk}$ from pre- to post-exposure phases for all sham-treated animals. Only the responses at test T_a from 26.6 to 36.6 °C are presented because these were the temperatures where the largest effects of temperature and microwave treatments were seen. It is apparent once again that mean values of $\overline{T}_{sk}$ tend to become more alike as the exposure phase advances.

As stated earlier, responses of T_{co} and M exhibited little change from baseline levels during the chronic exposure phase. Figure 14 shows the physiological responses of animals chronically exposed at 1 and 5 mW/cm^2 at test T_a=29.9 °C, the temperature which demonstrated the largest effects of microwave exposure. When combined with the results in Figure

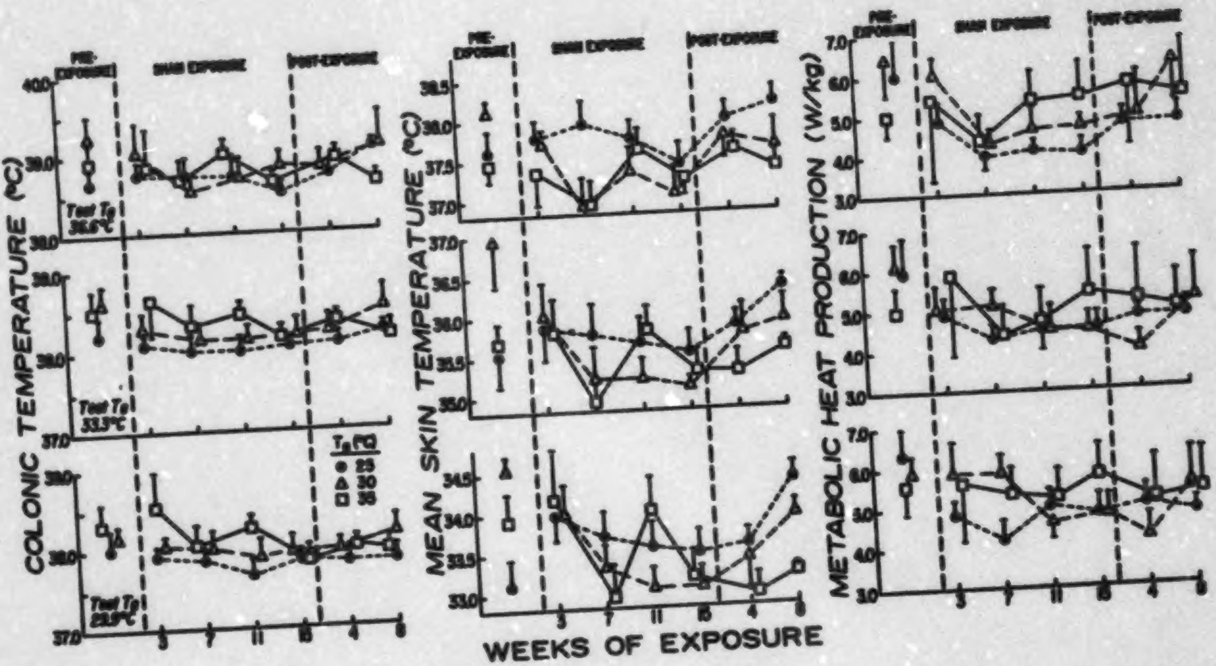

Figure 13. Colonic temperature, mean skin temperature and metabolic heat production (presented from left to right) for sham-exposed animals (n=4) as functions of exposure time. All values represent acute responses measured at test temperatures of 36.6, 33.3, and 29.9 °C (presented from top to bottom). Functions on each graph separate responses of animals chronically exposed to T_a of 25 °C (●), 30 °C (△) and 35 °C (□).

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13 it is clear that microwave exposure produced little change from the baseline levels of T_{co} and M. However, animals chronically-exposed to 30 or 35 °C and 1 mW/cm² (Fig. 14) exhibited a major increase in T_{co} and M during the post-exposure phase, whereas animals previously exposed at 5 mW/cm² at these ambient temperatures showed little change. Explanation of these results is difficult if one adopts the logic that with increased stress (e.g., microwave intensity) there should be an augmented change in any response which is casually related to the particular stress in question. Such is not the case in this situation.

Variance analysis indicated that the change in $\overline{T}_{sk}$ was affected by exposure to microwaves. Animals exposed at 1 mW/cm² in any of the 3 ambient temperatures showed a decrease in mean values of $\overline{T}_{sk}$ from a baseline level of 33.93 ± 0.32 °C to 32.78 ± 0.41 °C during test 2 of the chronic exposure phase (Fig. 14), with a similar decrease occurring in sham exposed animals (Fig. 13). Animals exposed to microwaves at 5 mW/cm² showed basically no change from a baseline level of 33.50±0.32 °C over the same time period (Fig. 14). Such differences may only reflect normal variations in this particular variable and may not be the product of temperature or microwave exposures. Statistical results which demonstrate a significant effect should be accepted with extreme caution in light of the small sample size used in this study and the extremely variable nature of the $\overline{T}_{sk}$ response.

Figures 13 and 14 both show the tendency for the metabolic response to decrease from its baseline level during the early stages of any chronic exposure used in this study. It is possible that this downward shift is due to a general decrease in resting metabolic rate (RMR), the result of either the particular treatments employed or simply generalized

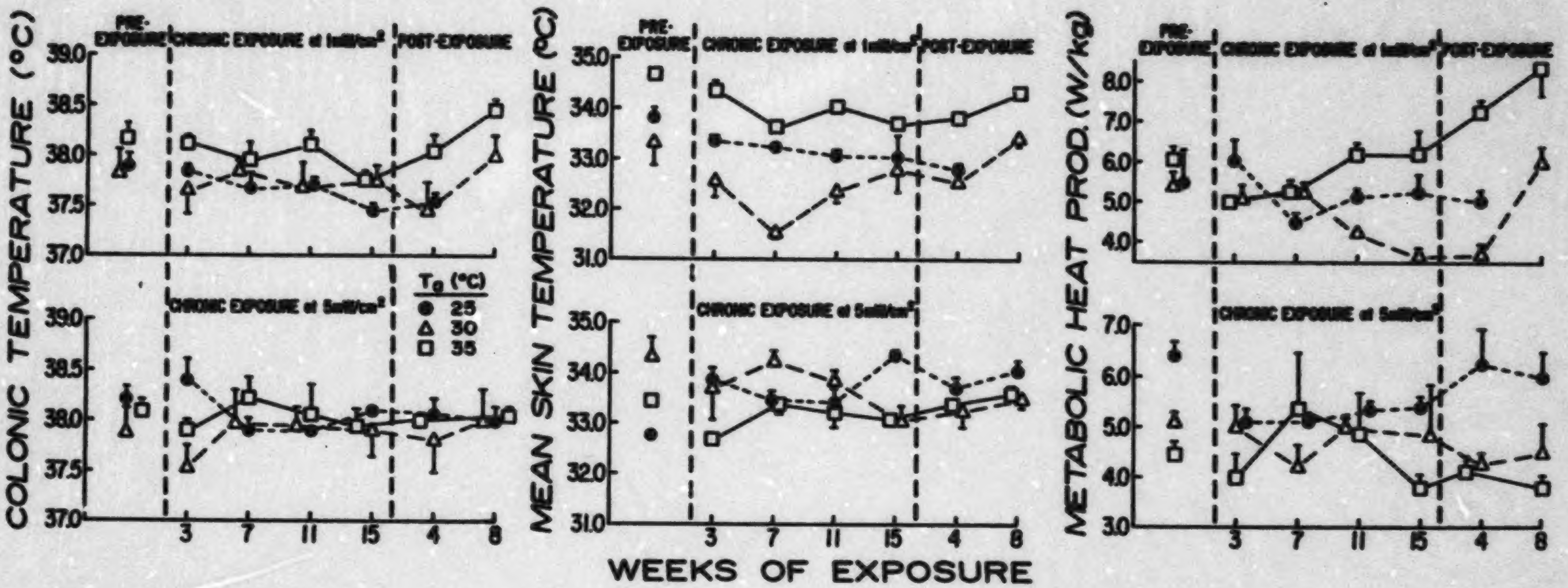

Figure 14. Colonic temperature, mean skin temperature and metabolic heat production (presented from left to right) at test T of 29.9 °C as functions of exposure time. Upper panels show responses of animals chronically exposed at 1 mW/cm^2; lower panels show responses of animals chronically exposed at 5 mW/cm^2. Functions on each graph separate responses of animals chronically exposed to T 25 °C (●), 30 °C (▲) and 35 °C (□). Sample size of each group was 2.

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adaptation to the test situation. The data for all animals were examined to determine if RMR changed during the exposure phase as a function of the conditions of chronic exposure. Resting metabolism was determined for each physiological test conducted on a given animal as the lowest measured value of M irrespective of test T_a. Variance analysis of mean baseline values of RMR showed no effect of either temperature or microwave exposure, implying that groups to be treated accordingly did not differ with regard to RMR. For each animal, the change in RMR (ΔRMR) during and after chronic exposure was determined by subtracting the mean baseline value from the response at the particular test in question. Variance analyses of ΔRMR during tests 2 and 4 of chronic exposure and test 1 of the post-exposure phase showed no significant effect of either temperature or microwave treatment on the response. In addition, no significant interaction was noted between the treatments.

Large intragroup variances were observed in both baseline and chronic exposure values of resting metabolism. A linear regression analysis was performed on the change in RMR with time as a function of baseline levels, to determine if a significant correlation existed between the magnitude of the pre-exposure RMR and the change that occurred during exposure. The results of the analysis showed a significant correlation ($r=0.80$) between pre-exposure values of RMR and the change in RMR at test 2 of the chronic exposure phase. The line of best fit is represented by the equation $Y=4.75-0.08X$, where Y is the change in RMR (W/kg) and X is the mean value of baseline RMR (W/kg). These results indicate that the change in RMR which occurred during the early portion of chronic exposure was not related to any particular treatment, but to pre-exposure values of RMR. Correlation coefficients for this relationship decreased with longer exposure, still being significant at 15 weeks (r=.053) but not at 5 weeks post-exposure (r=0.33). This indicates that pre-exposure levels of RMR are not as influential in determining values of RMR later in exposure, as in the early stages.

Each physiological test began with equilibration of the animal to a test T_a of 20 °C. Further analyses of the metabolic response were conducted to determine if the increase in metabolism during this acute cold exposure was altered by the conditions of chronic exposure. Resting metabolic rate was determined in each test and subtracted from the steady-state value of M measured at the initial test T_a of 20 °C. The difference was designated as the cold response for the particular animal at that test. Mean pre-exposure levels of cold response were calculated for each animal and an analysis of variance performed to determine if differences existed between groups before chronic exposure. No individual or interactive effects of temperature or microwave exposures were noted in baseline values. Variance analysis of the change in cold response from the pre-exposure level to tests 2 and 4 showed only a slight interactive effect of temperature and microwave treatments at test 2, with no significant effect of individual treatments at either time. These results indicate that the metabolic response to an acute cold stress was not greatly altered by chronic sham or microwave exposure at different temperatures within the limits of this study.

2) Sweat rate

Animals in all groups, except Group I, were monitored continuously during each physiological test to determine sweat rate ($\dot{m}_{sw}$). Responses were recorded during each test as shown in Figure 5 and analyzed (Figure

12) as a function of both T_{co} and $\overline{T}_{sk}$, each an important determinant of $\dot{m}_{sw}$. Slopes and Y-intercepts of the linear relationships between $\dot{m}_{sw}$ and both T_{co} and $\overline{T}_{sk}$ were calculated for each test. Only these values were then used for response comparisons. As for the other dependent variables, an analysis of variance was performed on the mean pre-exposure sweat responses of animals to be exposed to 0, 1, and 5 mW/cm^2 at 30 and 35 °C. Unfortunately, the absence of a record of Group I responses (T_a=25 °C at 0 and 1 mW/cm^2) did not allow a complete analysis of sweat rate at all exposure conditions. The results of the analysis indicated that the groups were not different before treatment.

Similar analyses were performed on the data collected during tests 2 and 4 of the chronic exposure phase using, in separate analyses, absolute and delta (chronic exposure - baseline) values. A significant effect of ambient temperature during chronic exposure on the relationship of $\dot{m}_{sw}$ to both T_{co} and $\overline{T}_{sk}$ occurred during test 2, but not test 4. The temperature effect is seen in Figure 15, which presents mean values of $\dot{m}_{sw}$ of all animals exposed to 25, 30, and 35 °C for 7 weeks, with no separation of data according to the intensity of microwave exposure. The slopes of the relationships of $\dot{m}_{sw}$ to T_{co} and $\overline{T}_{sk}$ are steeper for animals chronically exposed to T_a=35 °C. Such an effect indicates an augmented sweating response as a result of chronic heat exposure. A slight effect of microwave exposure on the response of $\dot{m}_{sw}$ to $\overline{T}_{sk}$ also occurred at 7 weeks of chronic exposure. A portion of this suggested effect is seen in Figure 16, which separates the results of chronic exposure to the

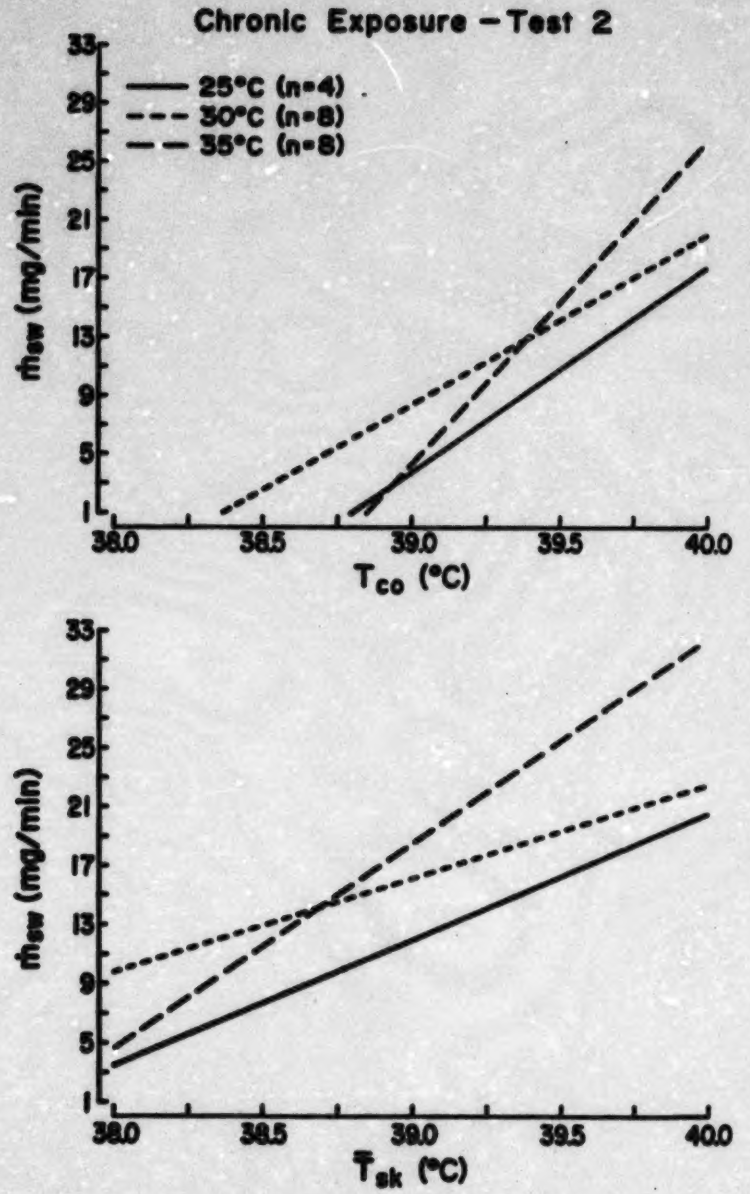

Figure 15. Sweat rate ($\dot{m}$) as a function of colonic temperature (upper graph) and mean skin temperature (lower graph). Data were collected during physiological test 2 of the chronic exposure phase. Each function represents mean values of linear characteristics (i.e., slope and Y-intercept) determined using values from all animals chronically exposed to T_a of 25, 30, and 35 ^{O}C, regardless of microwave treatment.

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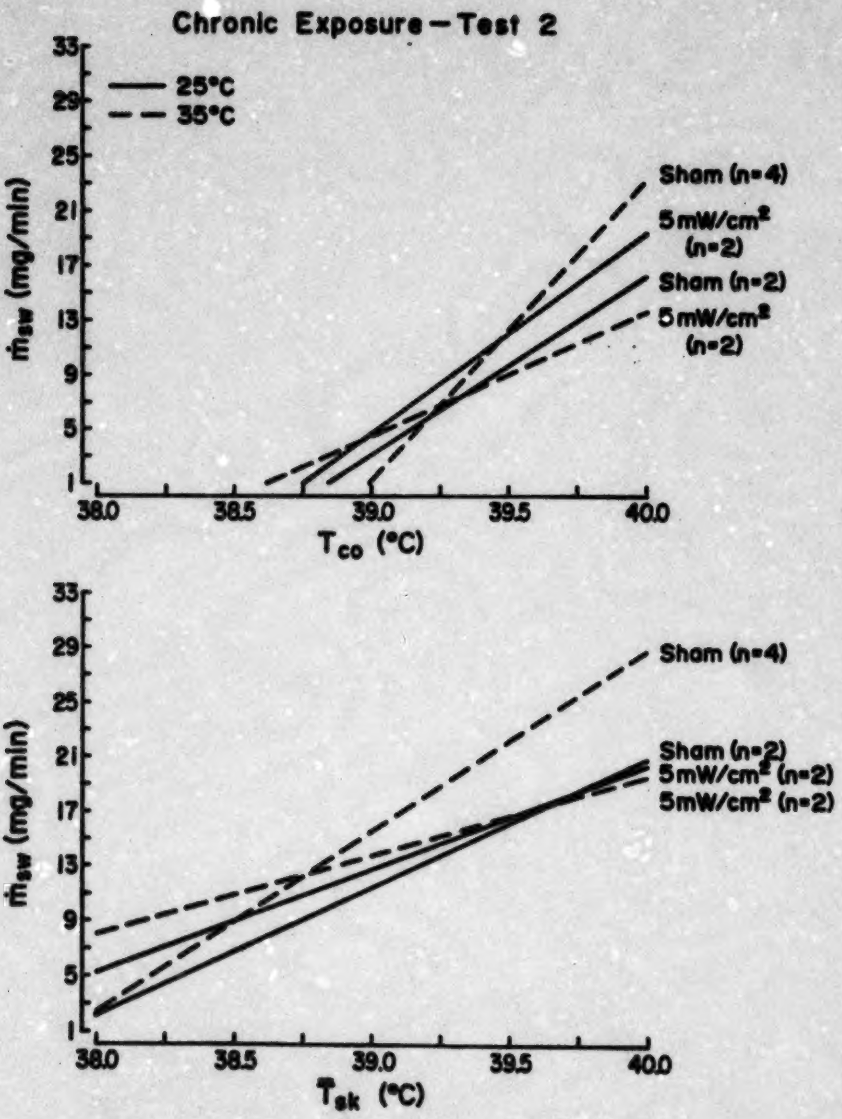

Figure 16. Sweat rate ($\dot{m}_{sw}$) of animals exposed to treatment extremes as a function of colonic temperature (upper graph) and mean skin temperature (lower graph). Data were collected during physiological test 2 of the chronic exposure phase. Each function represents mean values of linear characteristics (i.e., slope and Y-intercept) determined using values from all animals chronically exposed to combinations of T_a (25 and 30 °C) and microwaves (0 and 5 mW/cm^2).

extremes of microwave power density (0 and 5 mW/cm²) and T_a (25 and 35 °C) used in this study. In the relationship of $\dot{m}_{sw}$ to $\overline{T}_{sk}$, the slope of the function for sham-treated animals at chronic T_a=35 °C was greater than that for the 5 mW/cm²-exposed animals at the same T_a. Although this effect of microwave exposure is significant, it is unexplainable if one considers the fact that exposure to increased heat stress should increase, not diminish, the sweat response to an increase in either T_{co} or $\overline{T}_{sk}$. Interpretation of these results is further complicated by the outcome of an additional analysis of variance. Inclusion of the 25 °C response in such a comparison necessitates the removal of the 1 mW/cm² microwave response to balance the analysis in the absence of Group I data. With this comparison, no significant effects of either T_a or microwave exposure were seen at either test 2 or 4 during chronic exposure. Because of the contradictory nature of the ANOVA comparisons and considering the fact that the squirrel monkey has a poor and highly variable sweat response, it is questionable whether chronic temperature or microwave exposures, within the ranges explored in this study, had any "true" effect on this mechanism of heat loss. Further studies using larger population samples would be required to determine with certainty if the sweat rate of the squirrel monkey is altered by such treatments.

c. Behavioral Tests

1) Preferred Ambient Temperature

A total of 332 behavioral tests was conducted in the course of the study of which 72 were baseline tests of thermoregulatory behavior (no microwaves present) and the remainder were standard behavioral tests to determine the microwave threshold for the alteration of thermoregulatory behavior. For the most part, the data for each animal were analyzed separately; thus, each animal served as its own control. Means of T_{co} and $\bar{T}_{sk}$ and of the T_a selected by the animal were calculated across individual test sessions in 10-min time bins. Grand means and standard errors of each dependent variable were then calculated across both the 3 baseline tests and the 3 pre-exposure behavioral tests to serve as reference data against which the effects of chronic exposure might emerge. A sample of such baseline data for one monkey has been presented in Figure 7.

Preliminary examination of the behavioral tests conducted during and after chronic microwave and sham exposure indicated considerable variability in the behavioral response, both within individual animals and across animals in a given group. This was a natural consequence of the low frequency of testing (every 4 weeks) that was imposed by the experimental design. Refined operant responding of the type studied here can deteriorate markedly if not emitted frequently. Thus, in order to uncover trends in the data as a whole, considerable pooling and averaging of the test data were undertaken.

We first examined whether chronic microwave or sham exposure at different ambient temperatures altered the ambient temperature preferred by the animals. The pertinent data are the initial 90-minute baseline segments of each behavioral test when no microwaves were present (cf. Figure 7). Generally speaking, both thermoregulatory behavior and body temperatures had stabilized by the end of the first hour of the test; therefore, the mean T_a selected during the final 30 min of this baseline segment was calculated as the most accurate assessment of preferred T_a.

The data were then averaged for all the animals in each treatment group. Figure 17 (panel A) shows the mean T_a selected during the test baseline for all animals chronically-exposed at a T_a of 25 °C. This includes all animals in Groups I and IV. The points are plotted as a function of the week in which the test was conducted, measured from initiation of chronic exposure. Panel B shows the same data, now plotted in terms of the change in preferred T_a from the mean level during the pre-exposure phase. Sham-exposed animals and animals exposed to microwaves at 1 mW/cm^2 exhibited no change in preferred T_a either during the 15 weeks of chronic exposure or during the post-exposure follow-up period. However, the animals exposed to microwaves at 5 mW/cm^2 preferred a 1 to 2 °C cooler environment after the initiation of chronic exposure. Since this altered preference persisted during the post-exposure period, it is not clear that it was related to chronic microwave exposure per se. Indeed, it may only reflect a higher T_a selected by these animals relative to the other animals during the pre-exposure phase.

Similar analyses were performed on the remainder of the behavioral tests. The animals of Groups II and V, chronically-exposed at 30 °C, showed no shift in preferred T_a during and after the chronic exposure period. However, both the sham-exposed animals from Groups III and VI and those microwave-exposed at 5 mW/cm^2 from Group VI selected 1 to 3 °C cooler environments during all tests conducted during chronic exposure (at 35 °C), a trend that was reversed during the post-exposure period. Microwave-exposed animals from Group III exhibited a similar trend of lesser magnitude. Taken together, these results indicate that chronic

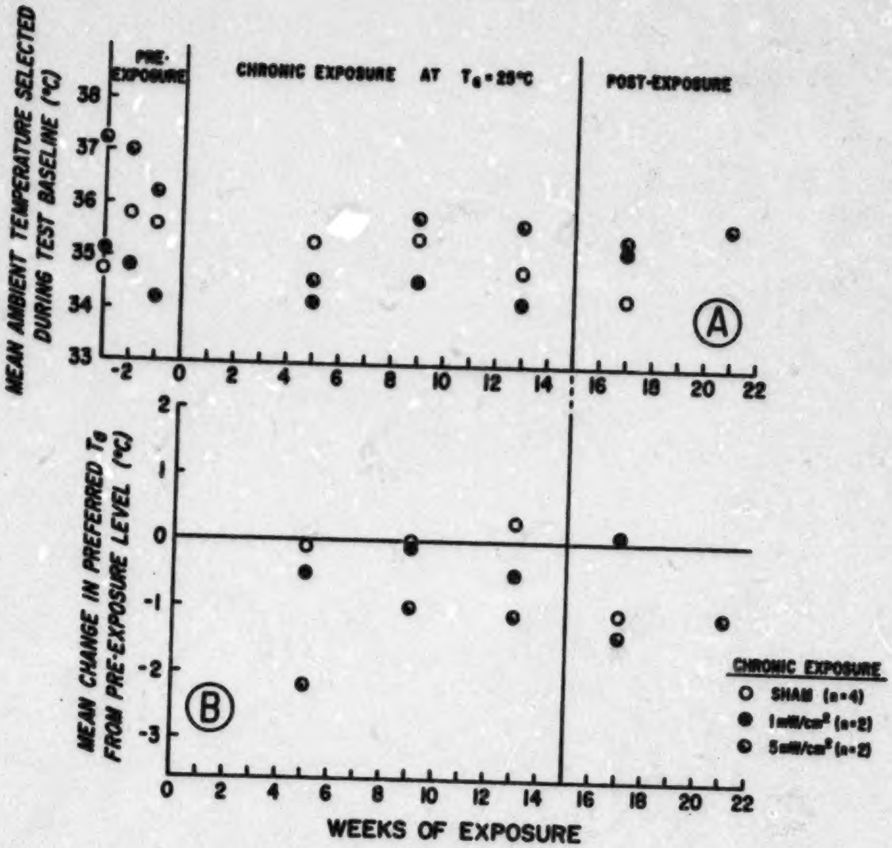

Figure 17. Mean ambient temperature selected during final 30 min of the behavioral test baseline by animals in Groups I and IV as a function of the week in which tests were conducted (panel A). Panel B shows mean change in preferred ambient temperature (T_a) during and after chronic exposure from level selected during pre-exposure period.

exposure to a warm environment (35 °C), with or without microwaves present, can stimulate squirrel monkeys to select somewhat cooler surroundings relative to their pre-exposure preference but that microwave exposure at the power densities employed usually does not provide comparable stimulation in cooler environments (25 and 30 °C).

2) Changes in Microwave Threshold

The criteria for assessment of the microwave threshold for the alteration of thermoregulatory behavior were stated above in the description of the behavioral test protocol (Methods) and illustrated in Figure 7. The major purpose of the behavioral tests was to determine if changes occurred in this threshold during the chronic exposure phase and whether any changes observed could be attributed to the specific exposure conditions employed. We first examined the data of each animal individually to determine the legitimacy of averaging the results from the three behavioral tests conducted at four-week intervals during the chronic-exposure phase. The analysis of the baseline data from these tests (Figure 17) had shown no significant shift in selected T_a across the 15 weeks of chronic exposure for any group. A similar test-to-test stability in thermoregulatory behavior during that portion of the test in which the animal was exposed for 10-min periods to 2450-MHz microwaves of increasing power density is illustrated in Figure 18, panel A. The mean selected T_a is plotted for each behavioral test as a function of experimental time for one animal from Group III-M (1 mW/cm^2 at T_a=35 °C). Although the average T_a selected during these tests was considerably below that selected during the baseline tests (shown by open symbols)

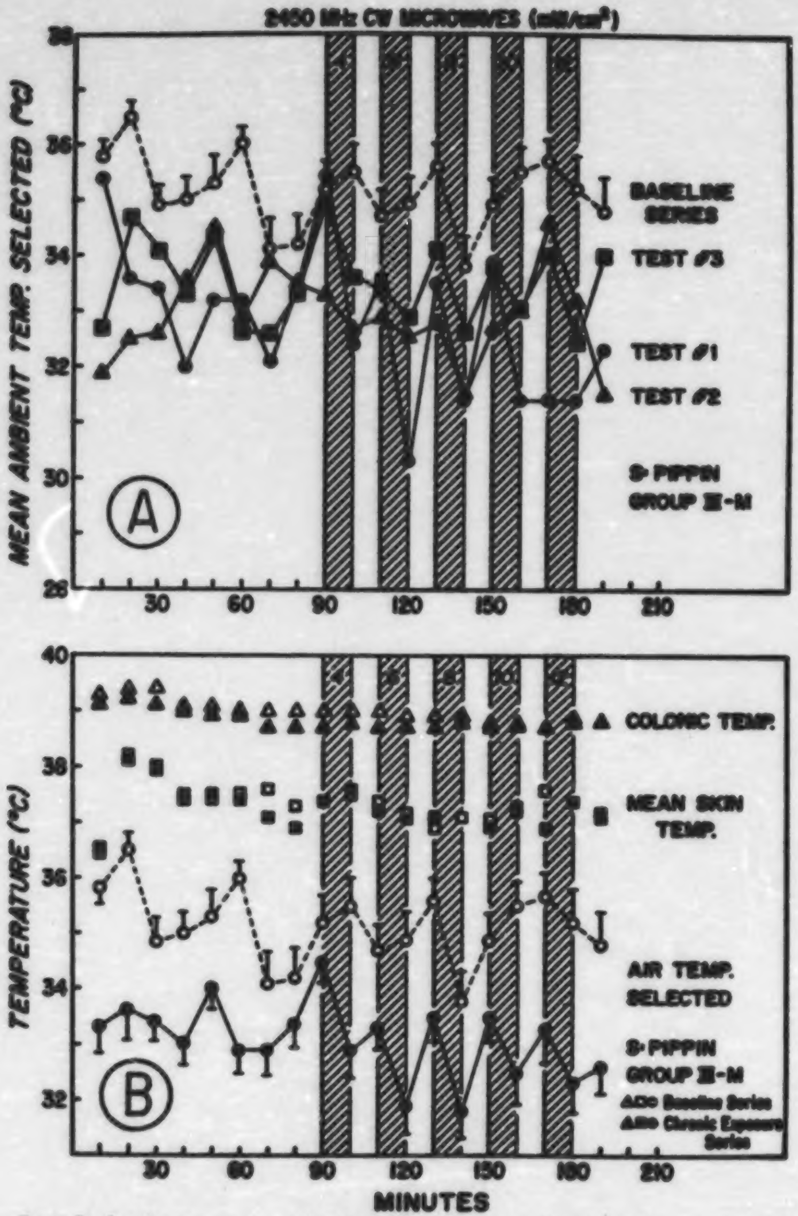

Figure 18. Panel A shows mean air temperature selected by one monkey during selected behavioral tests as a function of experimental time. Open symbols represent mean data (± 1 SEM) from 3 baseline tests during which no microwaves were present. Solid symbols represent data from behavioral tests 1, 2, and 3, conducted during chronic exposure, that involved five 10-min exposures to 2450-MHz microwaves at increasing power density (4,6,8,10, and 12 mW/cm^2). Mean colonic temperature, weighted mean skin temperature, and mean air temperature (± 1 SEM) selected by the same monkey during the baseline series (open symbols) and the chronic exposure series (closed symbols) are compared in Panel B.

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conducted prior to chronic exposure, it did not shift significantly from test to test as chronic exposure progressed. Inspection of the data from all animals yielded similar trends and provided justification for the averaging of test results across the chronic exposure phase. Panel B of Figure 18 shows the mean air temperature (±SEM) selected during these three behavioral tests as well as the T_{co} and $\overline{T}_{sk}$ produced by this behavior (solid symbols). Comparable data from the baseline tests are also shown (open symbols). These data should be contrasted with those in Figure 7, also for the same animal.

The microwave threshold for the alteration of thermoregulatory behavior prior to chronic exposure was 10 mW/cm^2 for this animal (Figure 7). However, the downward shift in selected T_a during chronic exposure renders difficult the application of the stated criteria for determination of the microwave threshold; nearly every point is different from the corresponding point during the baseline series. Normalization of the chronic-exposure function in terms of the average T_a selected during the initial 90 min of the tests allowed application of the threshold criteria and yielded a microwave threshold of 4 mW/cm^2 for this animal during chronic exposure, a clear reduction from the pre-exposure level.

Figure 19 shows comparable data for another (sham-exposed) animal from Group III; Panel A illustrates that the microwave threshold for the alteration of thermoregulatory behavior was 8 mW/cm^2 prior to chronic exposure at 35 °C. During chronic exposure, this animal also lowered its overall preferred T_a by several degrees. Normalization of the chronic-exposure function in terms of T_a selected during the initial 90-min of

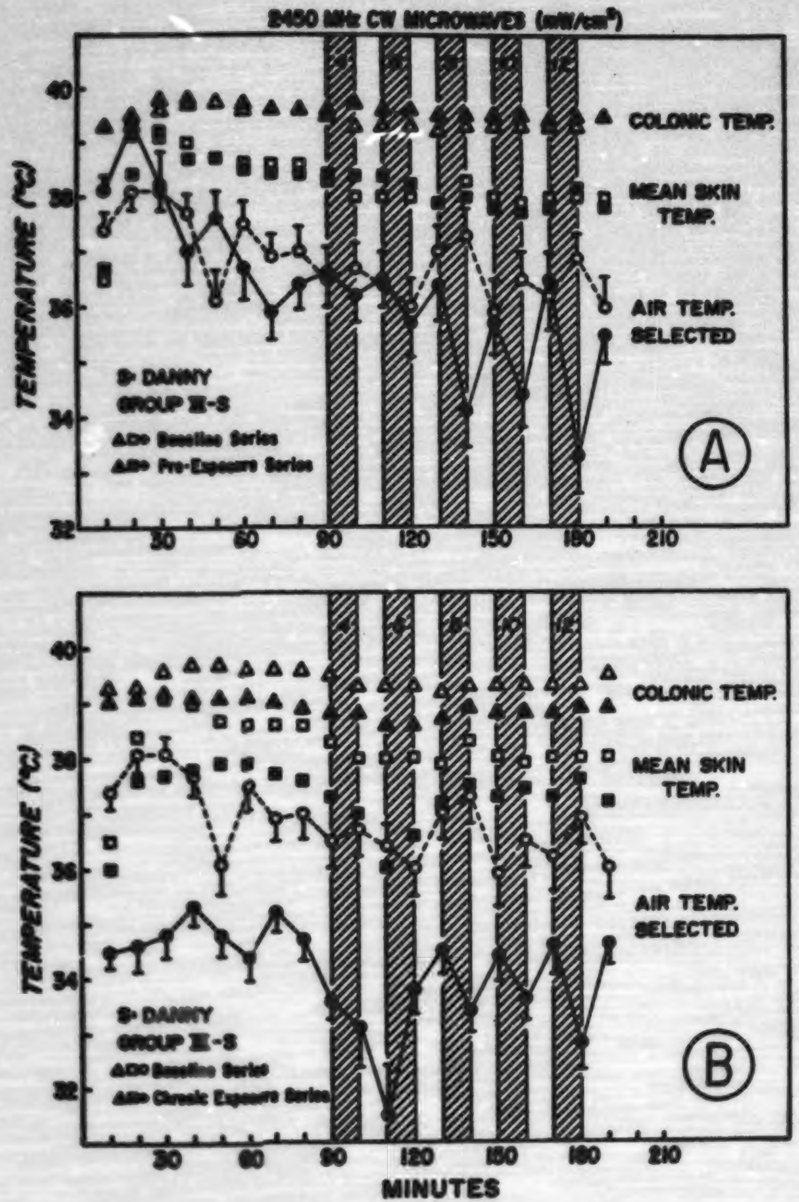

Figure 19. Panel A shows mean colonic temperature, weighted mean skin temperature, and air temperature (±1 SEM) selected in behavioral tests by one monkey as a function of experimental time. Open symbols represent mean data from 3 baseline tests during which no microwaves were present. Solid symbols represent mean data from 3 tests prior to chronic exposure that involved five 10-min exposures to 2450-MHz CW microwaves at increasing power density (4,6,8,10 and 12, mW/cm²). Panel B shows baseline data from Panel A (open symbols) now compared with mean data from the chronic exposure series (closed symbols).

the tests, prior to application of the threshold criteria, yielded an unchanged microwave threshold of 8 mW/cm^2 during chronic exposure.

These two examples delineate the major difficulty encountered during the analysis of the behavioral tests, a shift in the overall T_a selected across the period of testing which occupied approximately 5 months for each group. However, as stated above, the shift occurred randomly across treatment groups except for animals chronically-exposed at T_a=35 °C.

Shifts in T_a selected were not the only effects observed during the behavioral tests as the chronic exposure treatment progressed. Shifts in the regulated levels of T_{co} and $\overline{T}_{sk}$ were also evident for some animals, as illustrated in Figures 18 and 19. Although each of these animals selected a cooler environment during the chronic-exposure phase, normal body temperatures were maintained by S:Pippin (Figure 18) but not by S:Danny (Figure 19). Both T_{co} and $\overline{T}_{sk}$ were significantly lower in the latter animal during chronic exposure. This finding was judged to be random in that no other sham-exposed monkey in Groups III or VI exhibited a similar trend. Inspection of the mean data for all animals revealed no shifts in body temperature during or after chronic exposure that were correlated consistently with a particular set of exposure conditions. The number of animals in each group was too small to perform any statistical test to verify this conclusion.

The microwave thresholds for all animals during the pre-exposure, chronic exposure, and post-exposure series of tests are summarized in Figure 20. The panel on the left separates the data in terms of the T_a

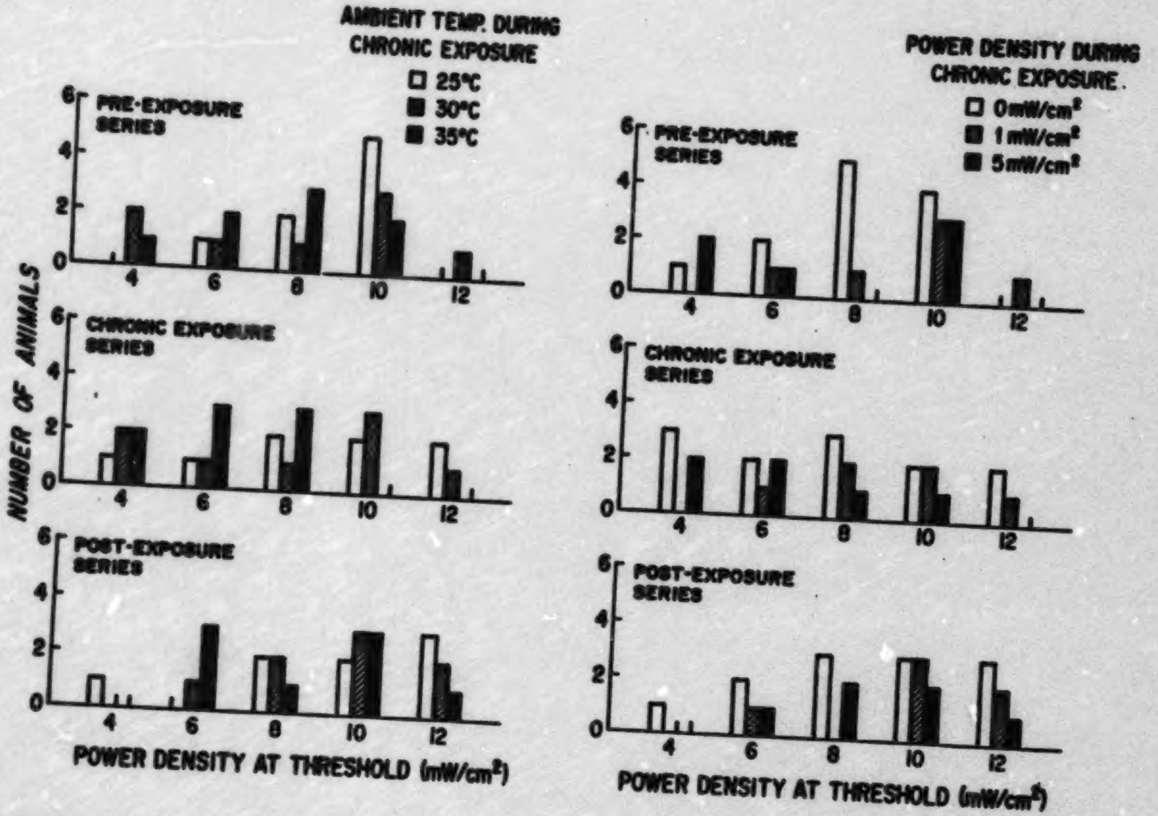

Figure 20. Distribution of microwave thresholds for the alteration of thermoregulatory behavior determined in behavioral tests during the pre-exposure, chronic exposure, and post-exposure phases of treatment. Left panel identifies thresholds in terms of ambient temperature (25, 30, 35 °C) prevailing during chronic exposure, irrespective of microwave treatment. Right panel identifies thresholds in terms of microwave power density (0,1,5 mW/cm^2) prevailing during chronic exposure, irrespective of ambient temperature.

prevailing during chronic exposure, regardless of sham or microwave exposure conditions; the panel on the right separates the data in the opposite manner. For each phase of treatment, the thresholds were distributed across the full range of power densities explored. These distributions are slightly more peaked during the pre-exposure series than in the other two and have an average value of 8-10 mW/cm^2, slightly higher than that reported by Adair and Adams (1980b). During the chronic-exposure phase, there is a tendency for the threshold to be slightly reduced, a tendency that was reversed during the post-exposure phase. This shift reflects primarily the response of animals chronically exposed at T_a=35 °C. However a two-way ANOVA revealed no significant effects of microwaves or ambient temperature alone nor of a combined effect ($p \leq 0.05$) on the microwave threshold for the alteration of thermoregulatory behavior.

d. Blood Tests

The results of the hematology tests performed on Groups I - III are presented in Table I. Mean values of white blood cell counts ranged from 5.4 x 10^3 to 12.1 x 10^3/µl. In most cases, there was a trend for a decrease in white blood cell count during the chronic exposure phase and a partial recovery during the post-exposure phase. Mean red blood cell count ranged from 6.2 x 10^6 to 8.0 x 10^6/µl. An increase in red cell count of 1.5 x 10^6/µl occurred from the baseline level to the level at week 10 of exposure in animals exposed to T_a=25 °C, with or without microwaves. At T_a=30 or 35 °C, the increase over the same time period was only 0.4 x 10^6/µl. Unfortunately, these results are difficult

Table I. Mean values of hematology measurements (±SE)

	Pre-Exposure	Chronic Exposure			Post-Exposure
	Week -1	Week 5	Week 10	Week 15	Week 20
White Blood Cell Count (x10^{3}/µl)					
25 °C					
Sham	12.1 (± 0.2)	9.1*	8.8 (± 1.1)	9.5 (± 0.6)	10.3 (± 0.2)
1mW/cm^{2}	10.4 (± 1.2)	10.0 (± 0.1)	11.5 (± 2.1)	9.5 (± 0.3)	7.6 (± 0.2)
30 °C					
Sham	8.6 (± 1.3)	6.9 (± 0.6)	7.0 (± 0.5)	6.3 (± 0.3)	8.3 (± 1.0)
1mW/cm^{2}	10.3 (± 0.2)	12.0 (± 2.6)	11.3 (± 1.0)	9.3 (± 0.3)	7.8 (± 1.3)
35 °C					
Sham	10.5 (± 1.1)	9.8 (± 0.3)	10.0 (± 0.1)	8.2 (± 0.8)	10.6 (± 0.6)
1mW/cm^{2}	6.1 (± 0.4)	5.4 (± 1.3)	5.9 (± 0.4)	7.1 (± 0.9)	7.7 (± 1.2)
Red Blood Cell Count (x10^{6}/µl)					
25 °C					
Sham	6.3 (± 0.4)	6.8*	7.9 (± 0.1)	7.2 (± 0.0)	7.5 (± 0.3)
1mW/cm^{2}	6.2 (± 0.1)	6.6 (± 0.1)	7.6 (± 0.3)	7.2 (± 0.0)	7.2 (± 0.0)
30 °C					
Sham	7.1 (± 0.3)	7.8 (± 0.3)	7.8 (± 0.3)	7.5 (± 0.1)	7.7 (± 0.4)
1mW/cm^{2}	7.5 (± 0.3)	7.3 (± 0.3)	7.5 (± 0.1)	7.3 (± 0.1)	7.6 (± 0.1)
35 °C					
Sham	6.7 (± 0.3)	7.4 (± 0.6)	7.0 (± 0.2)	7.2 (± 0.3)	7.5 (± 0.5)
1mW/cm^{2}	7.4 (± 1.2)	7.8 (± 0.4)	7.9 (± 0.6)	7.8 (± 0.4)	7.9 (± 0.6)
Hemoglobin (g/dl)					
25 °C					
Sham	12.0 (± 0.7)	12.7*	14.9 (± 0.1)	13.6 (± 0.1)	14.1 (± 0.1)
1mW/cm^{2}	12.3 (± 0.4)	13.3 (± 0.2)	15.8 (± 0.7)	14.8 (± 0.1)	14.4 (± 0.3)
30 °C					
Sham	15.0 (± 0.2)	16.7 (± 0.3)	16.2 (± 0.3)	15.6 (± 0.1)	16.2 (± 0.2)
1mW/cm^{2}	14.1 (± 0.1)	14.3 (± 0.4)	14.2 (± 0.3)	13.8 (± 0.2)	14.5 (± 0.0)
35 °C					
Sham	13.8 (± 0.3)	14.1 (± 1.0)	13.8 (± 0.5)	13.7 (± 0.2)	14.4 (± 1.1)
1mW/cm^{2}	16.6 (± 0.9)	16.5 (± 0.6)	16.3 (± 1.0)	16.2 (± 0.5)	16.4 (± 0.9)

Table I. Mean values of hematology measurements (±SE) (continued)

	Pre-Exposure	Chronic Exposure			Post-Exposure
	Week -1	Week 5	Week 10	Week 15	Week 20
Hematocrit (percent)					
25 °C					
Sham	33.9 (± 2.0)	34.8*	40.7 (± 0.8)	35.8 (± 0.6)	39.0 (± 0.2)
1mW/cm^2	34.4 (± 1.5)	37.3 (± 0.1)	43.2 (± 2.5)	40.2 (± 1.2)	39.7 (± 1.3)
30 °C					
Sham	42.4 (± 1.2)	47.3 (± 1.7)	46.7 (± 1.1)	43.3 (± 0.5)	45.7 (± 1.9)
1mW/cm^2	39.0 (± 0.2)	37.1 (± 0.6)	38.5 (± 1.3)	37.7 (± 1.2)	38.1 (± 0.5)
35 °C					
Sham	36.8 (± 0.9)	39.8 (± 2.8)	39.1 (± 1.4)	38.8 (± 0.2)	40.9 (± 2.3)
1mW/cm^2	44.4 (± 4.1)	46.7 (± 1.8)	47.2 (± 3.5)	46.1 (± 1.9)	48.2 (± 3.2)
Lymphocytes (percent)					
25 °C					
Sham	47.0 (± 3.0)	64.0*	50.0 (±10.0)	46.0 (±20.0)	62.0 (± 2.0)
1mW/cm^2	34.5 (±13.5)	37.0 (± 3.0)	34.5 (± 4.5)	34.5 (± 4.5)	51.0 (± 2.0)
30 °C					
Sham	38.5 (±19.5)	40.0 (± 1.0)	50.0 (±12.0)	38.5 (± 3.5)	57.5 (± 1.5)
1mW/cm^2	31.5 (±28.5)	45.0 (± 5.0)	49.0 (±15.0)	50.0 (± 6.0)	54.0 (±15.0)
35 °C					
Sham	34.0 (± 1.0)	44.5 (±13.5)	42.5 (± 1.5)	30.0 (± 3.0)	34.0 (± 6.0)
1mW/cm^2	51.0 (± 6.0)	49.5 (± 5.5)	35.0 (± 4.0)	33.5 (± 6.5)	32.5 (± 5.5)
Monocytes (percent)					
25 °C					
Sham	3.0 (± 1.0)	7.0*	5.0 (± 0.0)	4.5 (± 1.5)	3.5 (± 0.5)
1mW/cm^2	6.0 (± 1.0)	5.5 (± 2.5)	6.0 (± 3.0)	8.0 (± 0.0)	8.0 (± 1.0)
30 °C					
Sham	4.5 (± 2.5)	6.0 (± 2.0)	2.0 (± 0.0)	10.0 (± 1.0)	3.5 (± 2.5)
1mW/cm^2	3.5 (± 0.5)	1.0*	4.5 (± 2.5)	11.0 (± 1.0)	2.5 (± 1.5)
35 °C					
Sham	4.0 (± 2.0)	7.0 (± 2.0)	8.5 (± 2.5)	10.5 (± 3.5)	12.0 (± 2.0)
1mW/cm^2	5.0 (± 0.0)	5.0 (± 2.0)	6.0 (± 1.0)	5.0 (± 2.0)	7.0 (± 1.0)

* Only 1 sample

to interpret because the baseline counts of red blood cells at T_a=25 °C were less than at the other exposure temperatures. In fact, absolute values of red blood cell count at 5, 10, and 15 weeks of exposure were similar. Hematocrit values also increased by an average of 7.8% during the first 10 weeks of exposure to T_a=25 °C. The average increase at T_a=30 and 35 °C was only 1.9 and 2.6%, respectively. In most cases, hematocrit values increased during the exposure period and continued to increase during the post-exposure phase. Hematocrit values ranged from 33.9 to 47.3% across groups. Paralleling the increase in red blood cell count and hematocrit during chronic exposure was an increase in hemoglobin concentration. At T_a=25 °C, hemoglobin concentration increased an average of 3.2 g/dl during the first 10 weeks of chronic exposure, whereas the average increase at T_a=30 and 35 °C over the same time period was only 0.3 g/dl. Again, the large increase at T_a=25 °C may be related to the fact that the initial level at this temperature was lower than at any other chronic exposure temperature. The range of hemoglobin concentration was 12.0 to 16.7 g/dl. Values of percent lymphocytes were highly variable during the entire exposure period, ranging from 31.5 to 62.0%. Also, values of percent monocytes were not stable, ranging from 1.0 to 12.0%.

Blood electrolytes (Table II) (i.e., sodium, potassium, bicarbonate, chloride) did not exhibit any change during the chronic exposure phase which could be attributed to effects of temperature or microwaves. Average baseline values of sodium and potassium were 154.2 ± 1.5 meq/l

Table II. Mean values of serum chemistry measurements (±SE)
Blood electrolytes

	Pre-Exposure		Chronic Exposure		Post-Exposure
	Week -1	Week 5	Week 10	Week 15	Week 20
Sodium (meq/l)					
25 °C					
Sham	155.5 (±1.5)	154.5 (±0.5)	150.5 (±0.5)	154.0 (±1.0)	-
1mW/cm^2	149.5 (±2.5)	149.5 (±0.5)	154.0 (±5.0)	153.0 (±3.0)	150.0 (±2.0)
30 °C					
Sham	155.0 (±1.0)	158.0 (±6.0)	159.5 (±3.5)	156.5 (±1.5)	162.5 (±2.5)
1mW/cm^2	155.0 (±2.0)	153.5 (±1.5)	151.5 (±1.5)	152.0 (±1.0)	153.5 (±1.5)
35 °C					
Sham	150.5 (±1.5)	149.5 (±4.5)	147.5 (±1.5)	147.5 (±1.5)	148.5 (±1.5)
1mW/cm^2	159.5 (±0.5)	162.0 (±1.0)	160.0 (±8.0)	155.5 (±4.5)	157.5 (±9.5)
Potassium (meq/l)					
25 °C					
Sham	6.3 (±1.0)	6.0 (±0.2)	4.7 (±0.0)	6.2 (±0.5)	-
1mW/cm^2	5.3 (±0.8)	4.6 (±0.3)	7.0*	5.4 (±0.8)	4.6 (±0.2)
30 °C					
Sham	5.9 (±0.5)	6.7 (±1.3)	6.8 (±1.2)	6.4 (±0.9)	6.2 (±0.8)
1mW/cm^2	5.8 (±0.2)	6.0 (±0.4)	5.9 (±0.6)	5.6 (±0.6)	5.7 (±0.4)
35 °C					
Sham	5.4 (±0.1)	5.0 (±0.3)	4.8 (±0.2)	5.9 (±0.1)	5.3 (±0.5)
1mW/cm^2	6.7 (±1.4)	7.6 (±1.1)	4.8*	5.8 (±1.6)	6.5 (±2.0)
Bicarbonate (meq/l)					
25 °C					
Sham	7.9 (±2.7)	7.7 (±0.9)	17.4 (±1.1)	4.8 (±3.9)	-
1mW/cm^2	12.4 (±8.3)	18.2 (±2.2)	15.9 (±0.1)	9.7 (±1.8)	16.2 (±4.0)
30 °C					
Sham	11.7 (±5.2)	17.7 (±1.3)	7.3 (±0.6)	6.5 (±2.9)	11.3 (±0.1)
1mW/cm^2	5.9 (±0.9)	8.8 (±3.4)	6.2 (±1.2)	6.3 (±1.3)	6.9 (±1.9)
35 °C					
Sham	9.4 (±1.5)	8.9 (±0.8)	8.6 (±4.9)	10.1 (±0.2)	11.4 (±4.4)
1mW/cm^2	11.7 (±5.8)	6.3 (±1.3)	17.4*	11.4 (±7.7)	10.3 (±5.30)

Table II. Mean values of serum chemistry measurements (±SE)
Blood electrolytes (continued)

	Pre-Exposure	Chronic Exposure			Post-Exposure
	Week -1	Week 5	Week 10	Week 15	Week 20
Chloride (meq/l)					
25 °C					
Sham	121.0 (±0.0)	126.0 (±3.0)	116.0 (±1.0)	124.0 (±0.0)	-
1mW/cm²	117.5 (±4.5)	116.5 (±1.5)	115.0 (±0.0)	119.5 (±1.5)	114.5 (±1.50)
30 °C					
Sham	121.0 (±2.0)	119.5 (±4.5)	124.5 (±3.5)	124.0 (±4.0)	123.5 (±0.5)
1mW/cm²	119.0 (±0.0)	124.5 (±0.5)	122.5 (±4.5)	120.0 (±0.0)	117.5 (±1.5)
35 °C					
Sham	120.5 (±2.5)	118.0 (±1.0)	119.5 (±2.5)	124.0 (±2.0)	121.0 (±1.0)
1mW/cm²	122.0 (±4.0)	127.5 (±2.5)	118.0*	126.0 (±5.0)	122.5 (±4.5)

* Only 1 sample

- No sample

and 5.9 ± 0.2 meq/l, respectively. Bicarbonate and chloride levels were 9.8 ± 1.1 meq/l and 120.2 ± 0.7 meq/l, respectively.

Table III shows the values of thyroxine indices for Groups I-III. Upper and lower limits of serum concentration of thyroxine (T_4) were 6.4 and 0.8 μg/100 ml, respectively. Results of variance analysis performed on the change in T_4 concentration from baseline levels showed a significant effect of ambient temperature at 5, 10, and 15 weeks of chronic exposure. The major increase in T_4 concentration was at T_a=25 °C. Again, this result is difficult to interpret because the baseline concentration of T_4 at T_a=25 °C (1.5±0.8 μg/100 ml) was lower than at T_a=30 °C (3.1±0.8 μg/100 ml) or T_a=35 °C (4.3±1.3 μg/100 ml). Although the change in T_4 concentration was significantly affected by T_a, the absolute levels of T_4 at the end of the chronic exposure phase were not different. Likewise, no group differences were noted between absolute values of thyroxine binding capacity at the end of the chronic exposure phase. Once again, changes in this variable during the chronic exposure phase were related to initial baseline differences.

Serum concentrations of protein (Table III) did not exhibit any specific changes from the baseline level of 6.8±0.2 g/dl during chronic sham or microwave exposure at any T_a. Changes in serum levels of albumin from the mean baseline value of 3.6±0.1 g/dl were variable, with no apparent effect of either type of treatment.

DISCUSSION

A longitudinal study has examined whether the normal thermoregulatory responses of squirrel monkeys may be altered during and after

Table III. Mean values of serum chemistry measurements (±SE)
Thyroxine indices

	Pre-Exposure		Chronic Exposure		Post-Exposure
	Week -1	Week 5	Week 10	Week 15	Week 20
Thyroxine (μg/100 ml)					
25 °C					
Sham	0.8 (± 0.5)	3.1 (± 0.8)	2.1 (± 0.5)	2.9 (± 1.0)	1.7 (± 0.0)
1mW/cm^2	2.3 (± 1.5)	3.3 (± 0.6)	2.7 (± 1.2)	5.0 (± 1.0)	3.0 (± 1.0)
30 °C					
Sham	4.4 (± 0.1)	3.7 (± 0.5)	-	4.7 (± 0.3)	4.8 (± 0.7)
1mW/cm^2	1.7 (± 0.0)	0.9 (± 0.0)	0.9 (± 0.0)	0.7 (± 0.0)	1.3 (± 0.1)
35 °C					
Sham	2.2 (± 0.5)	2.4 (± 0.5)	2.4 (± 0.6)	-	2.0 (± 0.6)
1mW/cm^2	6.4 (± 0.8)	5.8 (± 0.7)	5.8 (± 1.0)	-	4.6 (± 0.4)
Thyroxine Binding Capacity (μg/100 ml)					
25 °C					
Sham	9.6 (± 1.2)	10.6 (± 1.9)	9.7 (± 0.5)	11.7 (± 0.4)	11.7 (± 0.4)
1mW/cm^2	10.7 (± 1.9)	11.1 (± 1.7)	11.8 (± 4.2)	12.5 (± 3.3)	13.6 (± 2.1)
30 °C					
Sham	16.9 (± 2.2)	19.9 (± 1.4)	-	16.2 (± 2.1)	10.7 (± 1.2)
1mW/cm^2	11.7 (± 0.4)	10.5 (± 1.5)	13.1 (± 0.6)	9.3 (± 1.2)	6.1 (± 0.2)
35 °C					
Sham	8.5 (± 0.4)	8.0 (± 0.5)	9.0 (± 0.1)	-	15.2 (± 1.2)
1mW/cm^2	12.6 (± 0.9)	12.4 (± 2.0)	13.8 (± 3.2)	-	20.7 (± 1.6)
Protein (g/dl)					
25 °C					
Sham	6.9 (± 0.2)	7.6 (± 0.5)	7.1 (± 0.1)	6.9 (± 0.1)	-
1mW/cm^2	7.6 (± 1.2)	7.3 (± 0.7)	7.4 (± 0.1)	6.6*	7.0 (± 0.9)
30 °C					
Sham	6.0 (± 0.2)	6.6 (± 0.2)	6.7 (± 0.2)	6.2 (± 0.2)	6.6 (± 0.0)
1mW/cm^2	6.7 (± 0.2)	6.8 (± 0.4)	6.6 (± 0.1)	6.3 (± 0.2)	7.0 (± 0.3)
35 °C					
Sham	7.3 (± 0.7)	6.4 (± 0.1)	6.1 (± 0.3)	6.6 (± 0.2)	6.4 (± 0.2)
1mW/cm^2	6.2 (± 0.1)	6.7 (± 0.2)	6.5 (± 0.7)	6.4 (± 0.3)	6.5 (± 0.5)

Table III. Mean values of hematology measurements (±SE)
Thyroxine indices (continued)

	Pre-Exposure Week -1	Chronic Exposure Week 5	Week 10	Week 15	Post-Exposure Week 20
Albumin (g/dl)					
25 °C					
Sham	3.6 (± 0.1)	3.9 (± 0.3)	4.2 (± 0.1)	3.6 (± 0.1)	-
1mW/cm²	3.3 (± 0.1)	3.6 (± 0.1)	4.1 (± 0.4)	3.5 (± 0.1)	3.7 (± 0.0)
30 °C					
Sham	3.7 (± 0.0)	4.2 (± 0.2)	3.7 (± 0.4)	3.4 (± 0.1)	3.8 (± 0.3)
1mW/cm²	4.2 (± 0.1)	4.3 (± 0.3)	3.4 (± 0.2)	3.5 (± 0.2)	3.8 (± 0.1)
35 °C					
Sham	3.4 (± 0.3)	3.1 (± 0.2)	3.1 (± 0.1)	3.0 (± 0.1)	3.2 (± 0.0)
1mW/cm²	3.6*	3.5 (± 0.1)	3.7 (± 0.7)	3.3 (± 0.3)	3.7 (± 0.4)

* Only 1 sample

- No sample

chronic exposure (40 h/wk for 15 wk) to 2450-MHz CW microwaves, at power densities of 0,1, and 5 mW/cm^2, when ambient temperature is simultaneously controlled at 25,30, or 35 °C. The findings were largely negative: no changes that occurred in metabolic heat production, resting metabolic rate, normal regulated body temperature, or the microwave threshold for the alteration of thermoregulatory behavior could be ascribed unequivocally to chronic microwave exposure. Chronic exposure to the 35 °C environment, irrespective of concomitant microwave exposure, increased sweating rate and lowered the environmental temperature selected by most animals. Significant shifts in mean skin temperature were difficult to interpret because of variability in baseline data, a difficulty compounded by the small sample size. No trends in the data over time indicated a cumulative influence of microwave exposure on thermoregulatory mechanisms. Tests of hematological and serum chemistry parameters yielded random results that were of dubious relevance to the exposure treatments. The most reliable finding appeared to be a reduction in body mass of the microwave-exposed animals regardless of exposure temperature, an effect that was most pronounced in those animals exposed at 5 mW/cm^2.

The study was exploratory, in the sense that a wide range of chronic exposure conditions was examined for possible thermoregulatory consequences. As a result, very few animals constituted each treatment group, rendering statistical analysis of the data very difficult. An alternative approach would have involved exposing larger numbers of animals to many fewer exposure conditions. However, no relevant experimental evidence existed on which to select the "critical" exposure conditions. In the belief that microwave exposure in thermoneutral and warm environments posed a greater threat to efficient thermoregulation than similar exposure in the cold, we selected exposure conditions that had the potential for heat stress, in the context of the thermoregulatory capacity of the squirrel monkey. Hence, the three ambient temperatures at which chronic exposure occurred were equivalent to the middle and two extremes of the thermoneutral zone of the species in question (Stitt and Hardy, 1971). The power densities selected were either well below (1 mW/cm^2) or near (5 mW/cm^2) the lower limit of those that can just provoke changes in thermoregulatory responses during acute microwave exposure under maximally-favorable environmental conditions (Adair and Adams, 1980 a, b; 1982). We believed that we had set the stage most favorably for the manifestation of cumulative effects and/or the enhancement of particular response changes to one environmental variable by a second variable acting synergistically. That most of the results were negative or equivocal reflects the influence of many factors, some of which are discussed below.

Acute vs chronic microwave exposure

It could be argued that since the monkeys spent less than 25% of their time in the chronic exposure environment, any alterations in thermoregulatory responses could have been reversed during the hours spent in the home cage. The 40 h/wk exposures were intended to simulate the temporal conditions in the workplace rather than those in the overall living environment. Studies of human subjects working in warm environments have demonstrated that acclimation to heat develops with as few as 2-4 h/day exposure for 7-9 days (Goldman, 1983). The fact that the animals in Groups III and VI (exposed to T_a=35 °C) preferred a cooler environment during the chronic-exposure phase provides counter-argument. This preference was well-developed in most animals by the first behavioral test, conducted in the 4th week of chronic exposure, and had begun to dissipate two weeks after chronic exposure had terminated. A change in thermoregulatory behavior of this type would be anticipated in animals undergoing acclimation to heat.

That concomitant exposure to microwaves, even at a power density of 5 mW/cm^2, did not enhance this behavioral effect is not particularly surprising; nor is the fact that chronic microwave exposure at lower T_a (25 and 30 °C) had no effect on thermoregulatory behavior. The higher power density employed (5 mW/cm^2) was below the threshold (6 to 8 mW/cm^2) determined by Adair and Adams (1981b) to alter this behavior and was well below the average microwave threshold (8 to 10 mW/cm^2) measured in the behavioral tests of the present study (Figure 20). A recent report by Adair and Adams (1983) concluded that subthreshold power densities would exert no influence over thermoregulatory behavior no matter how long the microwave exposure lasted. This conclusion implies no cumulative microwave effects, at least none of a thermal nature. An identical conclusion is inescapable in light of the results of the behavioral tests in the present study.

The 5 mW/cm^2 power density was also below the threshold level found to initiate most individual physiological thermoregulatory responses during acute microwave exposure (5 to 150 min) under the most favorable test conditions. No change in colonic temperature can be measured in animals exposed at this level in any T_a from 20 to 35 °C. A power density of at least 6 to 8 mW/cm^2 is required to initiate vasodilation of the tail vasculature in the squirrel monkey at a T_a (26 °C) just below that at which vasodilation normally occurs (Adair and Adams, 1980a). A power density of at least 6 to 8 mW/cm^2 is required to initiate thermoregulatory sweating from the foot of an animal at a T_a (35 °C) just below that at which sweating erupts spontaneously (Adair, 1981). Metabolic heat production appears to be slightly more sensitive to acute microwave exposure; a power density of 4 to 6 mW/cm^2 will reliably reduce the elevated heat production of monkeys in the cold (e.g. T_a=20 °C) (Adair and Adams, 1982). This range of power densities (4 to 8 mW/cm^2) represents a range of whole-body SAR from 0.6 to 1.2 W/kg, roughly 10 to 20% of the resting metabolic heat production of the squirrel monkey (Stitt and Hardy, 1971). In this context, it is not surprising that few response changes were observed to occur in animals chronically-exposed at 1 mW/cm^2 (SAR=0.16 W/kg). Indeed, most of the reliable effects observed (e.g. change in sweat rate, change in preferred ambient temperature) were shown to be related to the T_a prevailing during chronic exposure rather than to the presence or absence of microwaves.

No statistically-significant change occurred in the microwave threshold for the alteration of thermoregulatory behavior as a result of chronic exposure. This finding implies that long-term experience with low-level microwave fields probably confers no change in the thermal sensitivity to microwaves during acute exposures in other milieux. It was of interest to examine the thermoregulatory behavior of the animals for evidence of "irritability", a potential sequel of chronic environmental stress. Therefore, the continuous records of T_a selected during the behavioral tests were scrutinized for incidence of irregular response patterns and spuriously-high response rates during the 15-s reinforcement intervals when few or no responses should have been emitted. No extraordinary response patterns were detected for any animal. Further, throughout the course of the study, no animal showed unusual aggressive tendencies, failure to consume the daily food ration, or any evidence of illness of malaise. The reduction in body mass of all animals chronically-exposed to microwaves at 5 mW/cm^2 was the only physical change observed to occur in the presence of environmental conditions that were potentially stressful.

Changes in body mass

A reduced body mass in animals that are chronically exposed to microwaves is a finding at variance with those reported in other comparable studies of long-term effects. Usually no change in the body mass of microwave-exposed animals occurs relative to sham-exposed controls, although food intake may be depressed (Chou, et al., 1983; Ferri and Hagan, 1976; Lovely, et al., 1977, 1983; Moe, et al., 1976). Occasionally an increase (Djordjević, et al., 1977) or no change in the dependent variable (D'Andrea, et al., 1979) has been reported. The relative stability of body mass coupled with a reduced total food intake has been regarded as evidence for a metabolic compensation for absorbed microwave energy on the part of the exposed animals. It should be possible to test this hypothesis by measuring metabolic rate during the course of chronic exposure (Guy, et al., 1980).

Unfortunately, it is difficult to make precise measurements of the daily food and water intake of primates as can be done with small laboratory animals. Thus, there is no evidence that the loss of body mass in the monkeys chronically-exposed to microwaves at 5 mW/cm^2 was due to an alteration in food intake. It seems certain that no metabolic compensation was involved, however; no significant alteration in metabolic heat production was detected during the chronic exposure phase when the weight loss occurred. The range of individual decrements in body mass across the 6 affected monkeys was 11 to 95 g. Weight shifts of this magnitude often occur seasonally in male squirrel monkeys (Kaplan, _et al_., 1981; Middleton and Rosal, 1972; Colillas, _et al_., 1982) but no evidence for such seasonal shifts was uncovered in the analysis of the body mass data (Figure 10). The substantial increase in body mass (78.5±21 g) of the sham-exposed animals from Groups I and IV (Figure 11) is another curious finding. It is difficult to believe that this is a "true" effect of the experimental treatment since the environmental conditions for these animals during chronic exposure (25 °C) were little different from those prevailing in the vivarium. Replication of the study, using larger subject populations, will be necessary in order to evaluate isolated significant effects such as this for which no reasonable explanation can be found.

Changes in blood parameters

Values for white blood cell count, red blood cell count, hemoglobin, hematocrit, lymphocyte percentage and monocyte percentage have been reported for the squirrel monkey (Middleton and Rosal, 1972; Suzuki, 1981) and concentration values of sodium, chloride, potassium and total protein are known for the rhesus monkey (Rosenblum and Coulston, 1981). In most cases, the hematology values for the squirrel monkeys in this study were within the range of values reported as normal for adults. The only abnormal values were those for monocyte percentages, which in certain cases were unusually high near the end of the chronic exposure phase and during the post-exposure phase. No explanation can be given for these elevated concentrations.

Hematological changes during the chronic exposure phase were primarily limited to variables related to red cell numbers. Red blood cell count, hemoglobin and hematocrit values increased in both sham and microwave-exposed animals at T_a=25 °C. However absolute values for the red blood cell count and related parameters were not different across microwave exposures. The observed changes resulted from large variances in baseline levels. Changes in hematocrit have not been observed in squirrel monkeys exposed for 6 weeks to a severe cold (14 - 16 °C) or heat (36 - 39 °C) stress (Chaffee, et al., 1966). In addition, many blood properties remained unaltered in rats exposed to 2450-MHz microwaves at 5 mW/cm², 8 - 10 h/day, 5 - 7 days/wk for 16 weeks (D'Andrea, et al., 1979; Lovely, et al., 1983), indicating the absence of stress. Similar findings have been reported for rabbits (Chou, et al., 1983) exposed to 2450-MHz microwaves at 5 mW/cm², 35 h/wk for approximately 13 weeks.

Exposure of primates to a cold environment increases both the mass of the thyroid gland (Chaffee, et al., 1966) and the production rate of thyroxine (Gale, 1975) within 2 - 6 weeks of exposure. In addition, seasonal changes in serum levels of thyroxine have been observed in the male squirrel monkey (Kaack, et, al., 1980). Table III shows increased concentrations associated with chronic exposure to 25 °C. Again, this increase was primarily due to the low baseline values with no differences in absolute values attributable to sham or microwave exposure at any T_a.

Changes in Sweat Rate

It has been shown that sweating in humans is responsive to changes in both central (Benzinger, 1969; Nadel, et al., 1971) and peripheral (Banerjee, et al., 1969; Nadel, et al., 1971) temperatures. Active sweat glands in the rhesus monkey (Gisolfi, et al., 1982) respond similarly to human sweat glands during changes in both central and peripheral thermal inputs (Elizondo, et al., 1976; Smiles, et al., 1976). Evaporative heat loss, both respiratory and cutaneous, increases in the squirrel monkey in response to heat stress (Stitt and Hardy, 1971) and these responses may also be regulated by both central and peripheral inputs in this species (Stitt, et al., 1971). Unlike humans, sweating in the squirrel monkey is limited primarily to the volar surfaces of the hands and feet, as verified by both histological (Machida, et al., 1967) and physiological (Stitt and Hardy, 1971) evidence. In the physiological tests, low-level evaporation was recorded during the entire test period in all monkeys (Figure 5), with active sweating commencing near the end of the acute exposure to 36.6 °C or at a slightly higher ambient temperature. The upper limit of heat tolerance for the squirrel monkeys in this study falls within the tolerance interval of 35 - 39 °C noted by Stitt and Hardy (1971). They also reported that monkeys exposed to T_a=39 °C increased total evaporative heat loss to a level approximately 4 times that noted at T_a=30 °C. Therefore, sweating in the squirrel monkey appears to be an active thermoregulatory response, which is influenced by changes in ambient temperature above a certain threshold level. It remains to be determined if adaptive changes in sweating occur following long-term exposure to heat stress.

Acclimation to heat in humans reportedly involves an augmented production of sweat in response to input from both internal and peripheral thermosensitive sites (Chen and Elizondo, 1974; Mitchell, et al., 1976). In addition to the increased sweat production of heat acclimation, the latency for the onset of sweating is shortened and the threshold body temperature for the initiation of sweating is shifted to a lower level (Elizondo and Bullard, 1971). A similar response has been observed in sheep following heat acclimation (Hofmeyr, et al., 1969). Little information is available regarding acclimation to heat in primates. Smiles, et al. (1976) reported that warm-acclimated rhesus monkeys did alter sweat rate in response to changes in both central and peripheral temperatures; however, no comparisons were made with unacclimated monkeys to determine if the response was altered by acclimation. The thermal environment required to heat-acclimate the squirrel monkey is unknown. In the present study, monkeys were exposed 40 h/wk for 15 weeks to an ambient temperature as high as 35 °C in the presence of 5 mW/cm^2 microwaves. Variance analyses of the results provided evidence for and against acclimation to heat, depending on which groups were included in the analysis. In light of this contradiction, it is uncertain if the degree of thermal stress imposed on the animals was sufficient to produced heat acclimation. This is especially true considering the fact that the animals were not exposed over the 48-h weekend period each week, which could have resulted in some loss of any acclimation gained during the previous 120 hr.

Changes in skin temperature

Measurements of skin temperature have been used by numerous investigators as an indirect index of peripheral blood flow and, in steady-state conditions, to calculate thermal conductance from body core to skin. Several studies have quantified skin temperatures as a function of ambient temperature for the squirrel monkey (e.g. Stitt and Hardy, 1971; Lynch, et al., 1980). Peripheral vasodilation, determined as the point of rapid increase in local skin temperature, occurs in the tail and foot at ambient temperature ranges of 25.0 - 26.5 °C and 30.0 - 32.5 °C, respectively. No one, to our knowledge, has investigated the changes in skin temperature that may occur in primates as a result of long-term exposure to different ambient temperatures. In the present study, $\overline{T}_{sk}$ was analyzed to determine if baseline values at selected test T_a were altered following chronic microwave or sham exposure at different ambient temperatures. Findings have only been reported here for overall $\overline{T}_{sk}$ since detailed analysis of the data from specific skin sites will not be performed until a later date. In addition, no estimates of thermal conductance can be made because steady-state values are required for such an analysis; in the physiological tests, all measurements of $\overline{T}_{sk}$ at test T_a above 20 °C were transient responses.

Because skin temperature is highly dependent on blood flow to the skin site, responses tend to be extremely variable; this fact was evident from the significant differences seen between pre-exposure values of $\overline{T}_{sk}$ across the six groups. Baseline differences in any response measure before an experimental treatment has started make interpretation of the treatment-correlated response changes difficult and open to question. Significant effects of both temperature and microwave exposures on $\Delta\overline{T}_{sk}$ occurred at a test T_a of 29.9 °C during tests 2 and 4 of the chronic exposure phase. It is not surprising that exposure effects appear at this test T_a, since it is within the range of ambient temperatures that activate the vasodilatory response at various skin loci. At 29.9 °C, pre-exposure values of $\overline{T}_{sk}$ for animals to be exposed to 30 and 35 °C were not significantly different across microwave-exposure groups. However, $\overline{T}_{sk}$ at test 2 of the chronic exposure phase for sham- and 1 mW/cm^2-exposed animals was significantly depressed by at least 1 °C; animals exposed at 5 mW/cm^2 at these same temperatures did not show any significant change in this response. Although the changes were found to be significant, no logical explanation can be offered for the apparent shift upward in vasodilatory response in animals exposed to a lesser stress (i.e., 0 and 1 mW/cm^2). The absence of such a change in animals exposed to higher power densities of microwaves forces one to regard significant changes at lesser power densities with caution. Whether these differences are truly significant and reflect actual adjustments in vasomotor reflexes remains a question that can only be answered with additional studies using larger sample sizes.

Changes in colonic temperature and metabolic rate.

Colonic temperature and metabolic rate have been measured for the squirrel monkey (Stitt and Hardy, 1971; Adair and Adams, 1982) exposed acutely to ambient temperatures ranging from 20 to 35 °C . These variables were also measured in the physiological tests of the present study and found not to differ significantly from the earlier reports. Although the normal thermoregulatory responses of the squirrel monkey to different ambient temperatures are well described, there is no information regarding the adaptation of this species to either cold or hot environments. Indeed, there is limited information regarding the adaptive process for primates, with the exception of the human species, that may occur during chronic exposure to extremes in ambient temperature. Even with the abundant information for humans, the results are contradictory. Neither Joy (1963) nor Newman (1968, 1969) found any significant change in basal metabolic rate of men acclimated to intermittent cold exposures. However, Kang, et al. (1970) reported that Korean women divers exposed to cold water at regular intervals during the winter increased their basal metabolic rate by as much as 17%. Chaffee and Allen (1973) exposed rhesus monkeys to ambient temperatures of 5, 25 and 35 °C for 24 h/day over a period of 14 to 40 months. The RMRs of cold-acclimated monkeys were higher than those of either control or warm-acclimated animals; monkeys exposed to 35 °C experienced a reduction in RMR. In the present study, variance analyses were conducted using values of T_{co} of the chronic-exposure phase and compared with pre-exposure values. Neither ambient temperature nor microwave exposure had a significant effect on ΔT_{co}. A decrease in RMR from baseline occurred in many animals during the chronic-exposure phase. However, this decrease was primarily related to the level of RMR before chronic exposure began and was not attributable to any effects of the chronic exposure treatments.

Given no significant change in RMR following chronic microwave exposure, it is possible that the energy input was too low to produce any long-term effect. Since both metabolic rate and SAR were measured in this study, the percentage of normal metabolism that is represented by energy input via microwaves can be calculated. M was not measured at the exact exposure temperatures of 25 and 35 °C used for chronic exposure; however, it was determined at test T_a that were close to the exposure values. Estimates of M at 25 and 35 °C were 6.41 and 5.39 W/kg, respectively. If exposure at a power density of 5 mW/cm^2 constitutes an SAR of 0.75 W/kg, then energy inflow resulting from microwave exposure is equal to 11.7 and 13.9%, respectively, of total heat production by metabolism at these T_a. This indicates that internal heat production could be repeatedly altered by as much as 13.9% without producing any significant long-term changes in resting metabolism. Failure to exhibit a permanent change in RMR following microwave exposure may be attributed to one of two possibilities. The animals may not have responded metabolically to the additional energy load under these conditions but simply increased heat loss to maintain thermal balance. On the other hand, in light of the low microwave power densities used in this study, the animals may have only lowered M temporarily in response to the energy influx. One or both physiological adjustments would explain the lack of change in observed RMR.

Neither temperature nor microwave exposures significantly altered the metabolic response of monkeys to an acute cold stress (T_a=20 °C). A decreased metabolic response to acute cold stress has been shown in cold-acclimated humans (Wyndham and Loots, 1969). Lack of such a change in the monkey suggests either that they do not respond to long-term cold exposure as humans do or that exposure to 25 °C under the physical conditions we employed does not constitute a cold stress. The second possibility is the more likely.

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